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DISEASES OF ENT

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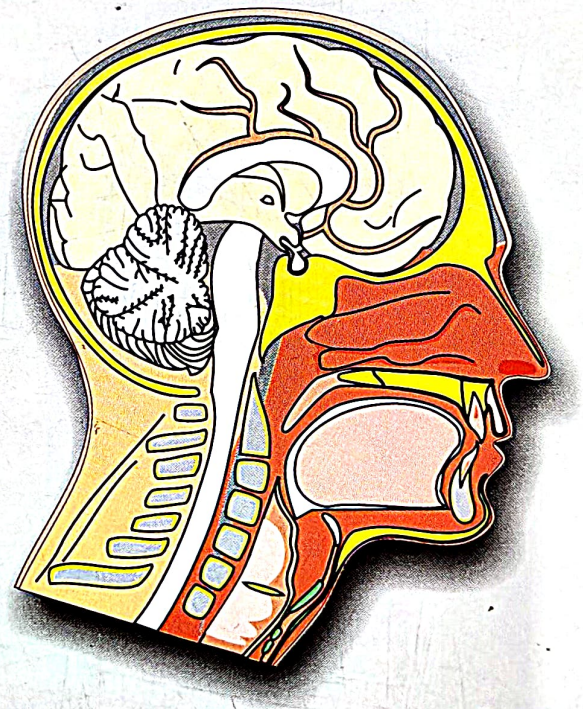

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SECTION - I

EAR

CHAPTER 1

Anatomy of the Ear

Anatomically the ear is divided as :

1) Pinna (Auricle)
2) External auditory canal
3) Middle ear
4) Inner ear

Pinna

The Pinna comprises of a single fibro-elastic cartilage covered by thin skin. This cartilage is continuous with that of the external auditory canal and extends throughout the entire pinna. The lower part of pinna is called lobule, which is a soft fibro-fatty connective tissue, covered by skin.

The Auricle has several prominences and depressions.

Helix is the outer curved border of the pinna, which starts at concha and ends at lobule. A small auricular tubercle referred to as Darwins tubercle is present on the postero-superior aspect of helix.

Anti- helix is a prominence, anterior to and parallel with the helix. Superiorly it divides into two crura, to form a triangular fossa. Superior to these crura lies the scaphoid fossa.

Concha is a cartilaginous part which lies anterior to anti-helix.

Cymba concha is a antero-superior descending part of helix, medial to it, lies Macewan's triangle in the temporal bone.

Tragus is a small blunt triangular prominence, situated anterior to the ear canal. It covers the canal orifice and is directed posteriorly.

Inter-tragic notch separates the tragus and anti-tragus.

Incisura terminalis is the area between the root of the helix and tragus which contains fibrous tissue and is devoid of cartilage (thus the site for Endaural incision).

The skin of posterior part of pinna is thin and devoid of hair follicles, but contains sebaceous glands. (site for Sebaceous cysts formation).

Concha and Scaphoid fossa have fine hairs with sebaceous glands. The perichondrium supplies the nutrients and remove the by-products of cartilage.

Pinna (External Ear)

Ligaments of Auricle

1) Anterior ligament - From tragus and anterior part of crus of helix to the root of zygomatic arch.
2) Posterior ligament - From posterior surface of pinna to mastoid.
3) Intrinsic ligaments - From anti-helix to helix.

Muscles - Attached to perichondrium.

Extrinsic muscles -

1) Auricular anterior
2) Auricular posterior
3) Auricular superior

They all insert into the aponeurosis of epicranium after radiating from pinna.

Intrinsic muscles (six) are vestigeal in man. Intrinsic muscles change the shape of auricle where as extrinsic muscles move the complete pinna in response to sound in animals.

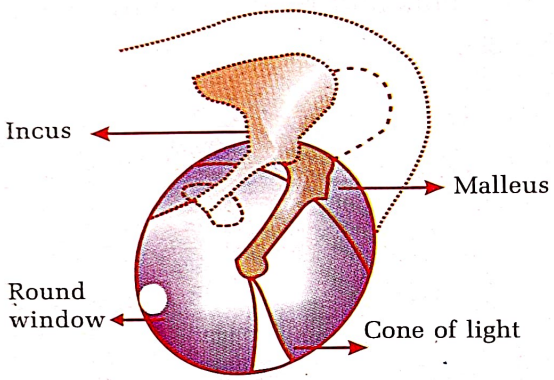
Blood supply

Anterior surface

- anterior auricular branches of superficial temporal artery branch of external carotid artery.

Posterior surface

- posterior auricular artery branch of external carotid artery.
- auricular branch from the occipital artery.



Right Tympanic membrane

Venous Drainage

- (follows arteries)
- Superficial temporal and posterior auricular veins

Lymphatics

Posterior surface	- Postauricular node (Mastoid tip)
Tragus, Anterior surface of upper part	- Preauricular nodes
Rest of Pinna	- Upper deep cervical nodes (Superficial cervical nodes)

Nerve supply

Laterally

Upper 2/3rd	- Auriculo-temporal nerve. (mandibular branch of Trigeminal nerve)
Lower 1/3rd	- Greater auricular nerve.

Medially

Upper 1/3rd	- Lessser occipital nerve. (C2, C3)
Lower 2/3 rd	- Greater auricular nerve.

Root of concha & part of medial surface

- Auricular branch of Vagus nerve.
- (Arnold's / Alderman's nerve)

Tragus and crus of helix

- Facial nerve along with auriculotemporal nerve.

Motor

Extrinsic muscles are supplied by Facial nerve.

External Auditory Canal (External Acoustic Meatus)

It extends from base of concha to the tympanic membrane. The length is about 2.5 cm and has a tortuous course, the outer part being cartilaginous and inner bony. The canal is therefore straightened by lifting the pinna *upwards and backwards* for visualization of the tympanic membrane. In children the manipulation of the pinna is done exactly opposite to that of the adults, and the pinna is pulled *downwards and backwards*. The characteristic of the canal skin is growth of epidermis, with shedding of keratin layers towards the ear canal.

The canal is oval in cross section. The greatest diameter is vertical at the lateral end and antero-posterior at the medial end.

In the *outer one-third* of the external ear canal, the cartilage of the pinna is continuous with that of the canal. It is adherent to the canal wall by fibrous bands. Fissures of Santorini are fissures found in this cartilaginous part, through which vessels and nerves pass. These are the source of transfer of infections from parotid or post auricular mastoid to ear canal or vice-versa.

The *inner two-third* of the canal is formed by the squamous and tympanic portions of the temporal bone. The skin of the canal is thin and firmly attached to periosteum. It is

continuous with that of the pinna i.e. the external auditory canal is the only skin lined cul-de-sac in the human body. The hair and sebaceous glands line the cartilaginous part of the canal as elsewhere in the body and also have ceruminous glands which lie in the deeper portions of the dermis.

The bony part is narrower than cartilaginous end i.e. it becomes smaller towards the tympanic membrane. The anterior wall is 4 mm longer than the posterior wall, due to obliquity of tympanic membrane.

The *Tympanic sulcus* is a groove at the medial end of canal in which lies the tympanic membrane. Annulus is a thick fibro-cartilaginous ring of tympanic membrane. Sulcus is absent superiorly (Notch of Rivinus), but contains an intrinsic ligament.

The *roof of the canal* is formed by squamous bone where as rest of canal is formed by the tympanic bone, i.e. tympanic sulcus. Due to this there are two suture lines in the canal i.e. anteriorly tympano-squamous and posteriorly tympano- mastoid suture line. (between the mastoid process and squamous part of tympanic bone).

Wax

Wax is a mixture of the secretory products of sebaceous and ceruminous glands. Wax contains a high concentration of lipids which accounts for its hydrophobic properties that provide a physical protective mechanism to the canal skin. The presence of lysozomes and immunoglobulins in wax are responsible for the defence systems existing in the external auditory canal.

CONSTRICTIONS

1) About 5 mm from the tympanic membrane, a narrow constriction called Isthmus occurs due to bulge of anterior canal wall. A wedge shaped space is present deep to this constriction, between the canal and tympanic membrane. It acts as a reservoir of infection, due to collection of pus and debris.

2) The second constriction is at the junction of bone and cartilaginous part.

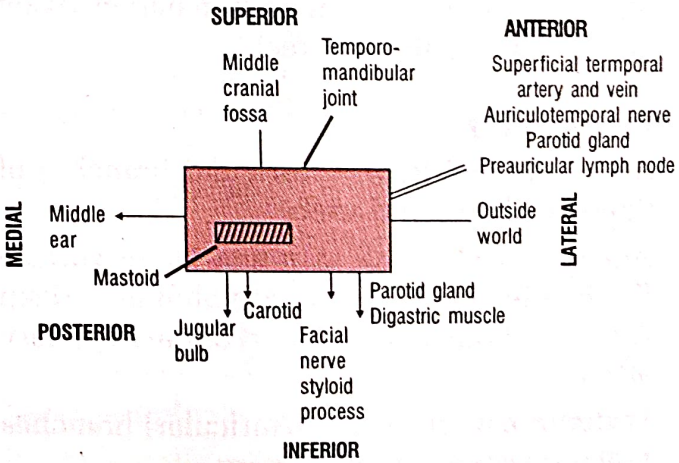
These constrictions are the common sites for impaction of foreign bodies.

Foramen of Huschke

This is an inferior channel formed by the fusion of bony growth of opposite spurs at the ventral aspect of bony canal by the age of 1 year. This smaller canal is located below the external auditory canal and closes later during childhood. Some times this may be a source of transfer of infections from parotid to ear canal or vice-versa.

Relations of EAC

Anteriorly	- Temporo-mandibular joint - Inner 2/3 rd Head of the mandible
Superficially	- Superficial Temporal vessels - Auriculotemporal nerve - Upper part of Parotid gland - Preauricular lymph node
Superiorly	- Middle cranial fossa
Posteriorly	- Mastoid air cells & postero-superiorly Antrum. Vertical part of Facial nerve deep to it
Inferiorly	- Parotid gland



Relationships of the right external auditory canal

Nerve Supply

Anterior ½ of meatus	-	Auriculotemporal nerve branch of Trigeminal nerve
Posterior ½	-	Auricular branch of Vagus nerve (Arnold's / Alderman's nerve)
Concha (sometimes posterior ½)	-	Nervus Intermedius OR Nerve of Wrisberg branch of Facial nerve

Arnold's Nerve (Alderman's Nerve)

It is formed by a large branch from Superior ganglion of Vagus (Xs) and from inferior ganglion branch of Glossopharyngeal nerve.

(Auricular branch of Vagus nerve)

Passes over the Jugular bulb

↓

bony channel to the Fallopian canal

↓

Superior branch — Inferior branch

Superior branch - the twigs of this branch ends in Facial nerve sheath.

Inferior branch - it supplies cutaneous sensation to posterior part of ear canal, after uniting with Facial nerve twig.

(the cutaneous eruption seen in herpes zoster oticus is due to these fibres)

Blood supply

Anteriorly and Roof – Auricular branches of Superficial temporal artery

Anterior meatal wall skin and Outer surface Epithelium of Tympanic Membrane – Deep Auricular branch of first part of the Maxillary artery

Posterior part of canal – (Auricular) branches of the posterior auricular artery.

Venous drainage – Superficial temporal and posterior - auricular veins which drain into the external jugular vein,the maxillary veins and pterygoid plexus.

Lymphatic drainage – Pre-auricular, post auricular & External jugular lymph nodes

Tympanic Membrane (Ear Drum)

The tympanic membrane is a grayish white (pearly grey) elliptical disc, separating the middle ear from the external auditory canal. In adults, it is placed obliquely at an angle of 55° with the floor of meatus. It faces downwards, forwards and laterally.The membrane is convex towards the tympanic cavity and its central point exhibiting maximum convexity is called the Umbo. (Knob on the center of a warrior's shield)

(R) Tympanic membrane

The Tympanic sulcus is a groove at the medial end of canal in which lies the tympanic membrane annulus, a thick fibro-cartilaginous ring of tympanic membrane. Sulcus is absent superiorly (Notch of Rivinus) but it contains an intrinsic ligament. This annular fibrous band from superior limit of sulcus runs centrally to the lateral process of malleus and its handle as anterior and posterior malleolar folds .

It is divided into

1) ***Pars Tensa :*** It is a lower, larger part of tightly stretched (tense) membrane. It is the area below the malleolar folds. It is divided into 4 quadrants by drawing an imaginary line from malleus handle to annulus and other imaginary line bisecting it at the level of umbo. The quadrants are named as Antero-superior, Antero-Inferior, Postero-Superior and Postero-Inferior.

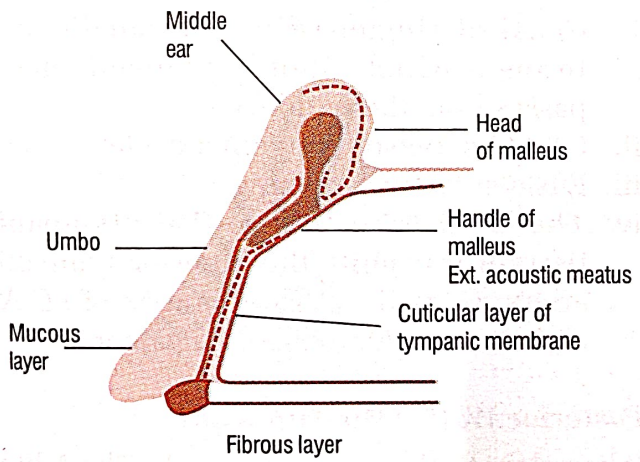
   In antero-inferior quadrant a 2 mm conical reflection of light is seen i.e.Cone of Light

2) ***Pars Flaccida*** (Shrapnell's membrane) : It is the area situated in between notch of Rivinus and malleolar folds, above the lateral process of malleus. It is pink in colour and flaccid. This loose membranous part is crossed by Chorda tympani in its inner surface.

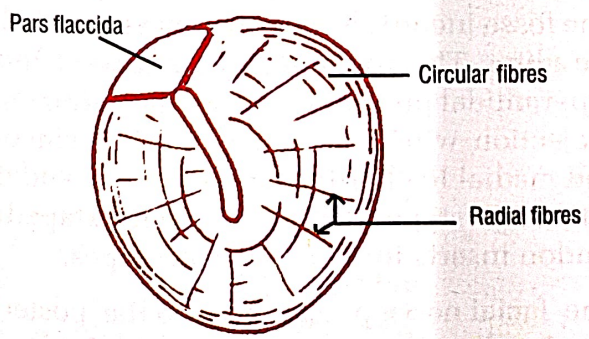
The surface area of is 80 mm^2. Its the membrane thickness is 0.1 mm. The horizontal & vertical diameter are 8-9 mm and 9 - 10 mm respectively. (weight 14 mgm). It is firmly held by the inward pull of the tensor tympani muscle, which is inserted into the upper part of malleus handle. Its elasticity is close to that of rubber.

The layers are :

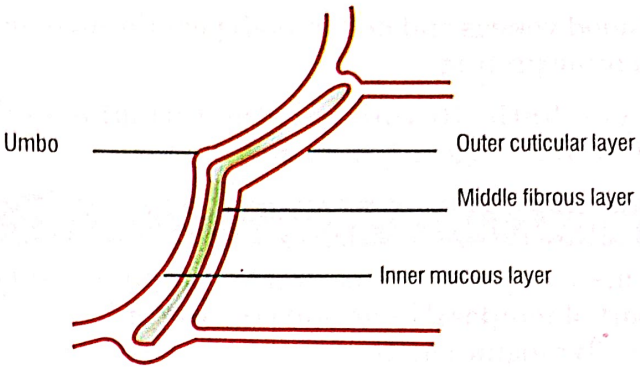
1) *Outer epithelial layer* – is continuous with the epithelium of the external auditory canal. (Cuticular layer).
2) *Middle fibrous layer* – This layer consists of outer radial and inner circular, parabolic and transverse fibres. It is very thin in the pars flaccida (loose areolar tisssue) and encloses malleus handle.



Section of Tympanic membrane



Arrangement of tympanic membrane fibres



Curvatures of the tympanic membrane

3) *Inner mucosal layer* – It is a continuation of middle ear mucosa. (Low ciliated columnar epithelium)

Blood supply :

Internally:

1. Arteria nutrica malleo-incudei – branch of Middle menigeal artery. supplies the Pars flaccida.
2. The vascular circle at the periphery is formed by –
   i) Anterior tympanic artery – a branch of internal maxillary artery
   ii) Posterior tympanic artery – a branch of stylomastoid artery
   iii) Twig from the ascending pharyngeal artery (inferior tympanic artery)

Externally :

i) Deep auricular artery- a branch of 1st part of Maxillary artery.
ii) Arteria manubrii.

Venous Drainage :

Outer surface - External jugular vein.
Inner surface - Transverse sinus, dural veins and venous plexus surrounding Eustachian tube.

Nerve supply :

i) Externally – from the auriculo temporal nerve (Vc) in its anterior half and auricular nerve [i.e. Alderman's / Arnold's nerve (X)] in its posterior half.
ii) Internally – from the tympanic plexus.

Blood vessels and nerves both pass through the lamina propria.

Lymphatic Drainage : Pre-auricular and Retropharyngeal lymph nodes.

MIDDLE EAR CLEFT

This narrow cavity is situated in the petrous part of temporal bone and consists of

1) Tympanic cavity,
2) Eustachian tube,
3) Aditus to the antrum and
4) Mastoid air cells.

It is the part in between the external and inner ear.

The middle ear cleft is lined by mucous membrane and is filled with air.

The Tympanic cavity (Tympanum / Middle ear cavity)

The tympanum is an air filled irregular shaped cavity, that lies between tympanic membrane and inner ear. This cavity contains the Ossicles and their muscles.

The anteroposterior and vertical diameters are both about 15 mm. The transverse distance from roof is 6 mm, 2 mm in the center and 4 mm the near the floor .

It has a shape of a box having a

1) Anterior wall
2) Medial wall
3) Posterior wall
4) Lateral wall
5) Roof (Superior wall)
6) Floor (Jugular wall / Inferior wall)

Superior wall (Roof of the Tympanic cavity)

The roof is formed by tegmen tympani, which separates the middle ear from middle cranial fossa. Tegmen tympani is a thin plate of tympanic bone formed by petrous and squamous part . It extends posteriorly to form the roof of aditus and antrum.

It is separated from the mastoid antrum by the tegmen antri.

Persisting petrosquamous suture line may be a pathway for spread of infection, to the meninges. (KÖRNER'S SEPTUM)

Floor (Jugular wall)

Floor is formed by a thin plate of bone which separates the middle ear from the jugular bulb. Jugular bulb is the continuation of the sigmoid sinus and beginning point of internal jugular vein. Some times it may be deficient (congenital) and covered by fibrous tissue and mucous membrane, therefore exposing the bulb to the middle ear.

The tympanic branch of glossopharyngeal nerve enters the middle ear, near the medial wall of floor from its origin from the skull.

Anterior wall (Carotid wall)

It has a thin plate of bone which separates the cavity from the Internal Carotid artery.

It shows following openings.

i. Canal of Hugier (Anterior canaliculus) through which Chorda tympani nerve passes from the middle ear.
ii. Canal for Tensor tympani muscle
iii. Eustachian tube opening
iv. The Glasserian fissure (Petrotympanic fissure) transmits the anterior tympanic artery (Maxillary artery branch of I.C.A.) and the anterior malleolar ligament.

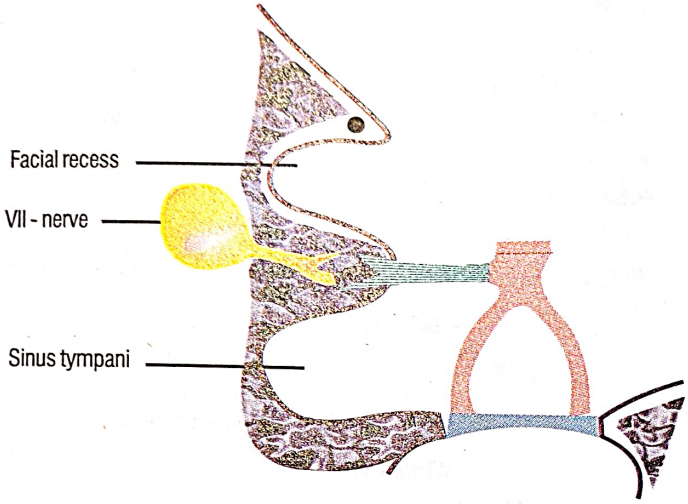
Posterior Wall (Mastoid wall)

It lies close to the mastoid air cells. The Aditus ad antrum is an opening in the superior aspect which communicates to the mastoid antrum. The fossa incudis is a small depresssion below the aditus. This contains short process of Incus. A pyramidal process is a small, hollow conical projection which lies below the fossa incudis and medial to chorda tympani nerve and this transmits stapedius muscle. The stapedius tendon inserts into the neck of stapes.

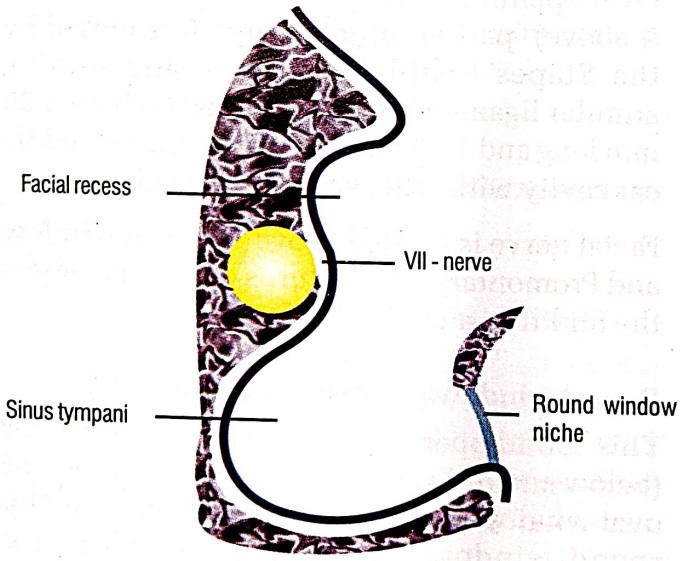
The facial nerve passes deep to the posterior canal wall, at the pyramidal process.

Facial recess [Suprapyramidal recess]

It is a space between the pyramid and tympanic annulus (lateral to the facial nerve). It is bounded medially by the facial nerve and laterally by the tympanic annulus. In between these, chorda tympani nerve runs medial to the membrane. A bony tympanomastoid septum separates the recess from mastoid air cells. This is approached directly in Posterior tympanotomy.



Sinus tympani and deep facial recess at the level of pyramid



Shallow facial recess at the level of round window

Lateral wall (Membranous wall)

This part separates the middle ear from external ear and is formed by the tympanic membrane, tympanic annulus and a thin outer attic /lateral attic/ epitympanic wall i.e. Scutum.

Medial wall (Labyrinthine wall)

This wall separates the middle ear from the internal ear. It is formed by the Labyrinth

The features are

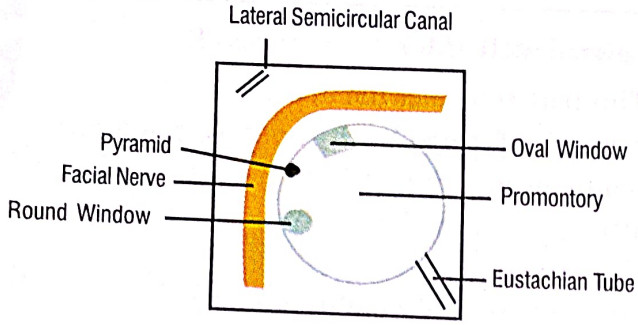
- Promontory (Basal turn of cochlea)
- Fenestra Vestibuli / F. ovale. (Oval window)
- Fenestra cochleae/F.rotunda (Round window)
- Processus Cochleariformis
- Sinus tympani.

Promontary : It is a smooth rounded bony projection of basal turn of cochlea. It occupies central part of medial wall and is covered by the tympanic plexus of nerves. Anteriorly it merges with the anterior wall of tympanic cavity. The facial nerve canal runs above it.

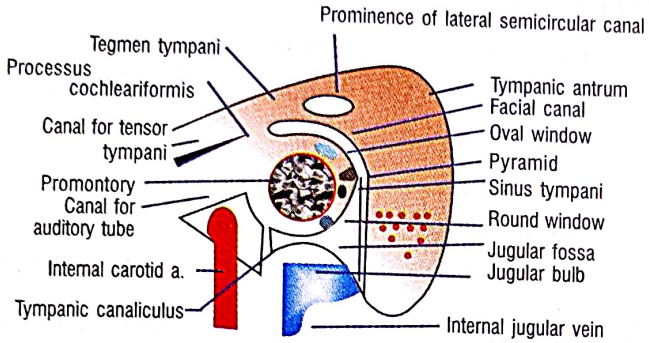
Subiculum is an posterior extention of the promontory (this separates oval window from round window).

Ponticulus is an bony spicule of promontory which passes to the pyramid on the posterior wall of cavity.

Ponticulus and subiculum divides the medial wall of posterior part of tympanic cavity into 3 depressions. The upper depression i.e. superior to the ponticulus is the oval window area, the lower depression i.e. inferior to the subiculum is round window area, and the middle area between these two ridges is Sinus tympani. (Tympanic recess).



Medial wall of the Middle Ear



Landmarks of the medial wall of the middle ear.

SINUS TYMPANI : This deep recess is located in between posterior and medial walls, medial to the pyramid.

Superiorly – limited by the Lateral semicircular canal and Ponticulus

Inferiorly – Subiculum
Styloid eminence
Jugular wall

Medially - Labyrinth

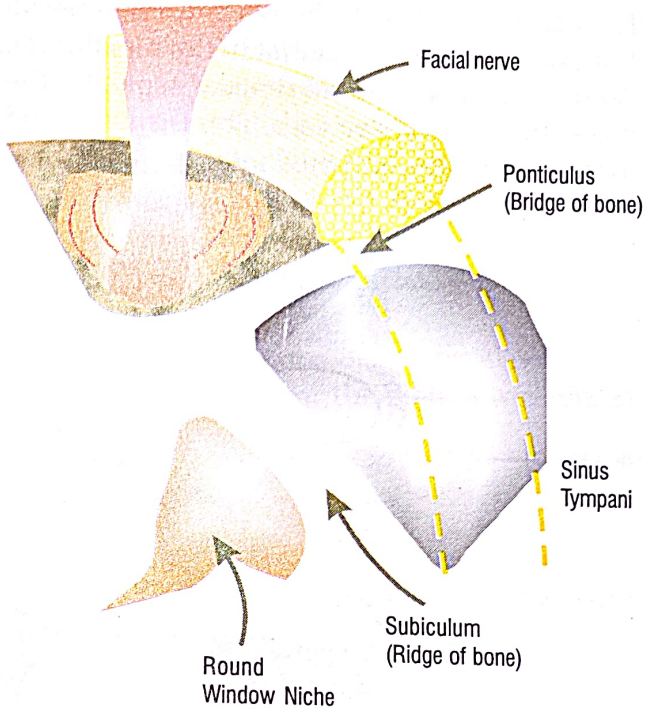
Laterally - pyramid process.
Facial nerve.

Sinus tympani lies deep to the facial recess.

This space is difficult to see with usual surgical approaches. Many times granulations and cholesteatoma will be missed in this space, which can be cleared by canal wall down technique.

Its medial wall continues as a medial wall of posterior part of middle ear cavity.

Two fenestrae - Round window niche (Below the subiculum)
Oval window niche (Above the ponticulus)



Relation of Ponticulus Subiculum

Oval window (Fenestra Vestibuli)

Oval opening in the postero-superior (behind & above) part of promontory. It is closed by the Stapes footplate with its surrounding annular ligament. Its average size is about 3.25 mm long and 1.75 mm wide. It connects middle ear cavity with scala vestibuli of cochlea.

Facial nerve is located above the oval window and Promontary. Here it turns down and enters the middle ear cavity.

Round window (Fenestra cochlea)

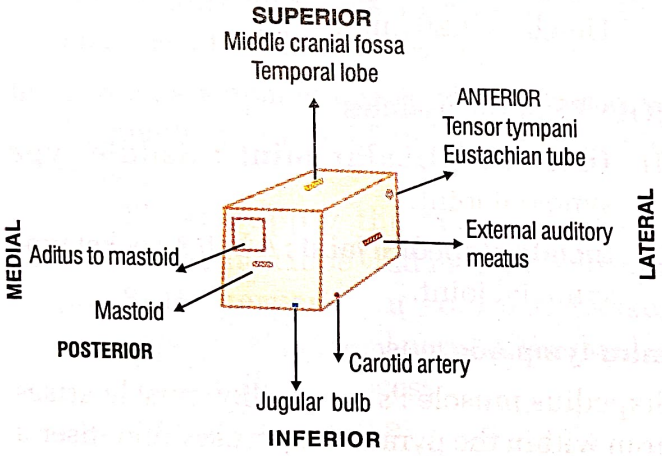
This round opening located postero-inferior (below and little behind) to the promontory (or oval window). It covers the scala tympani by round window membrane (i.e. secondary tympanic membrane). This membrane consists of an outer mucosal, a middle fibrous and an inner mesothelial layer.

The normal intact tympanic membrane protects it from the direct effects of loud sound. Normally the round window is not visible, due to over hanging edge of promontory.

The posterior ampulla of semicircular canal and singular nerve (Posterior Ampulary Nerve) is located 1 mm deep to the round window. This is the surgical land mark for the resection of singular nerve in the treatment of BPPV.

(Size - Anterior 1.5 mm, Superior - 1.3 mm, Posterior - 1.6 mm) (Area 2 mm^2)

Processus Cochleariformis : It is a hook like projection, anterior to the oval window. Here, the tendon of tensor tympani muscle takes a turn and inserts into the neck of malleus. The horizontal portion of facial nerve lies above this process. This forms an land mark for facial nerve surgery.



The relationships of the right middle ear

Compartments of the middle ear

Middle ear is divided into 3 compartments.

1) **Epitympanum (Attic)** : The part of the middle ear cavity that lies above the Pars Tensa but medial to the Pars flaccida and outer attic wall. (i.e. Scutum). This contains head of Malleus and body of incus.

2) **Meso-tympanum** : Lies medial to the Pars tensa. It contains stapes, long process of malleus, incus, oval window and round window.

3) **Hypotympanum** : This part lies medial to the and below the level of Pars tensa.

COMMUNICATIONS

Anteriorly - Eustachian tube connects this to Nasopharynx.

Postero-superiorly - Aditus connects to Mastoid antrum.

Contents

1) Ossicles : Malleus, Incus, Stapes
2) Intra -Tympanic muscles : Tensor tympani, Stapedius
3) Nerves : Chorda tympani nerve, Tympanic plexus
4) Ligaments of the ear ossicles
5) Vessels supplying and draining the middle ear.

Ossicles

MALLEUS : (hammer)

This is a largest ossicle.
It has got

- Head
- Neck
- Processes - Anterior process
- Lateral process (short)
- Handle

Head – rounded & lies in the attic. The saddle shaped postero- Medial surface articulates with incus.

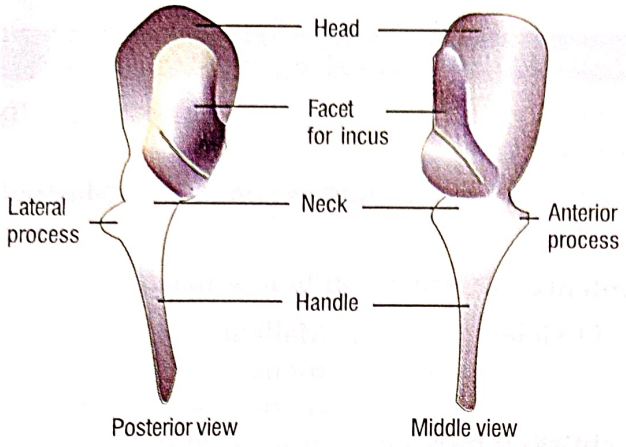
Neck – is the constricted middle portion.

Anterior process – it is a small anterior projection from which anterior ligament arises and inserts into the petrotympanic fissure.

Lateral process – is a projection from the upper end of handle which receives attachment from both malleolar folds.

Handle/Manubrium - is directed downwards and slightly backwards and firmly attached to the upper half of tympanic membrane. The tensor tympani tendon is inserted into the upper end of medial surface.

Size - 7.5 to 9.0 mm
Weight - 23 mgm



The malleus

Incus (Anvil)

Parts - Body
Processes - Short process
Long process

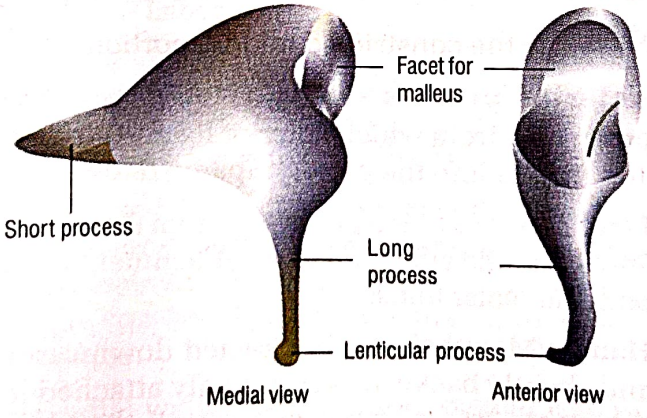
Body – lies in the epitympanumn. Its facet articulates with the malleus head.

Short process – lie in the fossa incudis and attached by a short ligament. It is projected backwards.

Long process – projects downwards just behind handle of malleus.

Lenticular process is a medially projected tip of incus, which articulates with stapes head.

Size 6 mm - 6 mm
Weight 25 - 30 mgm (average wt 27 mgm)



Left incus

Stapes

Smallest, medially placed bone of the ear.

i. Head
ii. Neck
iii. Cruras
iv. Base of Footplate

i. **Head** – Has a cartilage covered concave facet, which articulates with the lenticular process of incus.

ii. **Neck** – Stapedius tendon is inserted into the posterior part of narrow neck.

iii. **Cruras** – Arise from lower part of neck, both cruras join the convex.

iv. **Footplate of stapes** – It covers the oval window and is surrounded by annular ligament.

Weight - 2.86 mgm
Height - 3.26 mm

JOINTS of the ossicles

1) **Incudo-malleolar joint :** Saddle type synovial joint.
2) **Incudo-stapedial joint :** A ball & socket type synovial joint.

Intra-tympanic muscles

Stapedius muscle : short, bulky muscle arises from within the pyramidal process and inserts into the stapes. It is supplied by a branch of facial nerve.

The tensor tympani muscle

This bipennate slender muscle, arises from bony and cartilaginous part of eustachian tube and sphenoid and inserts into the upper part of malleus handle. It is supplied by the branch of the Mandibular nerve. (V)

This muscle tenses the tympanic membrane, where as stapedius dampens the loud noise.

Nerves : a. Chorda tympani nerve
b. Tympanic plexus

a. Chorda tympani nerve :

A branch of facial nerve. It enters the middle ear cavity from the posterior wall and runs medial to the tympanic membrane and upper portion of handle of malleus. It comes out through the canal of Huguier from the anterior wall and joins lingual nerve. The sensory fibres supply anterior 2/3rd of tongue. The parasympathetic fibres supply the submandibular and sublingual salivary glands.

b. Tympanic plexus :

The Tympanic plexus lies over the promontary It is formed by the

I) tympanic branch of the Glossopharyngeal nerve (Jacobson's nerve - arises from the inferior ganglion of 9th nerve and enters the middle ear cavity, through the inferior canaliculus and runs in a canal on the medial wall of the middle ear) and

II) Carotico-tympanic nerves which arise from Sympathetic fibres of the Internal carotid artery.
   i. These supply the tympanic cleft, Eustachian tube and mastoid antrum.
   ii. Both nerves join to form Lesser superficial petrosal nerve at the level of cochleariform process.
   iii. Twig of facial nerve

Blood supply of middle ear

Upper part of tympanic cavity

- Branch middle meningeal artery.
- Superficial petrosal artery.
- Superior tympanic artery.
- Ramus nutricia incudomallei.

Lower part – Inferior tympanic artery (Br. of Ascending pharyngeal artery)

Anterior part – Anterior tympanic artery (Br. of internal maxillary artery)

- The ramus-tympanici (a fine branch of internal carotid artery)

Posterior part – Posterior tympanic artery [Branch of mastoid artery → branch of stylomastoid artery → derived from posterior auricular artery. (fallopian canal)

Venous drainage

Drain into the superior petrosal sinus and the pterygoid plexus of veins.

Lymphatic drainage

Pre-auricular and retro-auricular lymph nodes.

Prussak's space

This small space lies between the pars flaccida (Sharpnell's) and the neck of malleus. It is bounded below by the lateral process of malleus and above by the fibers of lateral malleolar fold. The posterior gap communicates with the attic.

Mucosa of middle ear cavity

A thin delicate mucous membrane of the Eustachian tube and mastoid antrum is continuous to cover the tympanic cavity, and is reflected on the ossicles and intra-tympanic muscles. Thus formed folds, separate the middle ear into compartments.

The mucous membrane consists layer of single nonciliated cuboidal epithelium.

Anterior and inferior part of the tympanic cavity is lined by ciliated columnar epithelium where as cuboidal epithelium covers the posterior part.

EUSTACHIAN TUBE (Pharyngo-tympanic tube)

It has been named after Bartolomeus Eustachian from Rome. This 3.75 cm tube connects the middle ear to the nasopharynx. This serves mainly to ventilate, protect, and drain the middle ear.

The direction of tube in adults is upwards, backwards and outwards from the middle ear.

The tube is shorter, wider and more horizontal in infants and this anatomical variation results in frequent attacks of otits media following upper respiratory tract infections. The tube is lined by respiratory mucosa.

The lateral 1/3rd is bony extention of the middle ear cavity while the medial 2/3rd is the fibro-cartilaginous portion.

The bony portion runs in the squamous and petrous parts of the temporal bone. The Internal Carotid artery lies medial to this part of tube. The roof is formed by thin bone which is covered by tensor tympani muscle. This part is lined by ciliated columnar epithelium with mucous glands in the submucosa .

The anterolateral wall of medial 2/3rd is membranous (which contain Ostmann's lateral fat pad) and bent forward, while postero-medial part is formed by cartilage plate. This cartilage is fixed to the base of skull and is lined by pseudo-stratified ciliated columnar epithelium.

Isthmus is the angulated narrow part of the tube, where both portions meet.

The lower triangular opening lies at the level of the posterior end of inferior turbinate which is surrounded above and behind by Tubal elevation (wider medial end of tube).

This tubal elevation is pulled downards to the pharyngeal wall by salpingopharyngeal fold. The Levator palati forms a small swelling just below this opening, before entering soft palate. The fossa of Rosenmuller or Pharyngeal recess lies behind this tubal elevation. These are surrounded by lymphoid tissue/lymph nodes.

Muscles Attached to the Tube

1) **Tensor Palati :** (Tensor veli palatini)

   Origin :
   i. Bony wall of Scaphoid
   ii. Spine of Sphenoid
   iii. Along the whole/complete length of short/small superolateralpart of tubal cartilage.

   Insertion : This passes medially around the pterygoid humulus like a pulley and then meets the fibres of opposite aponeurosis within soft palate.

   This muscle separates the tube from following structures.
   1) Otic ganglion
   2) Mandibular nerve and its branches
   3) The chorda tympani nerve
   4) Middle meningeal artery

   Nerve supply : Mandibular nerve.

2) **Salphingopharyngeus :** This slender muscle is attached to lower part of tubal cartilage, near tubal elevation. It mixes/meets with palatopharyngeus.

3) **Levator palati :** (Levator veli palatini)

   Origin :
   i) Inferior part of Eustachian tube.
   ii) Inferior surface of petrous temporal bone.
   iii) Upper part of Carotid sheath fascia.

   Insertion : This enters the pharynx by passing over Superior constrictor and then spreads into the soft palate.
   Nerve supply : Both supplied by Pharyngeal plexus.

Action :

The tubal opening mechanism is poorly understood. The tensor palati is the only muscle which has a function in the opening of the Eustachian tube.

Blood supply :
1) Ascending pharyngeal artery
2) Middle meningeal artery

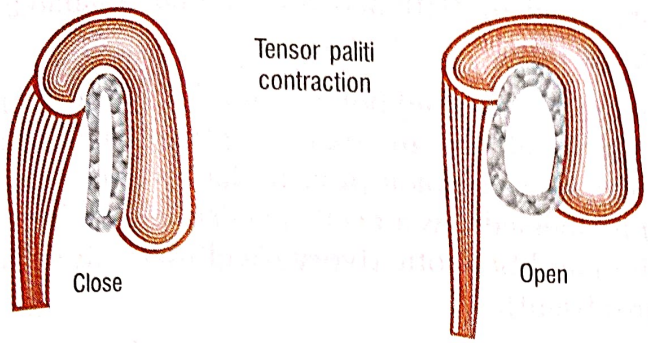
Venous supply : Drain into Pharyngeal plexus

Lymph nodes : Retropharyngeal lymph nodes

Nerve supply :

1) Ostium – The pharyngeal branch of the sphenopalatine ganglion (Vb).

2) Bony part – Tympanic plexus (Xc)
3) Cartilaginous part – Nervus Spinosus (Vc)



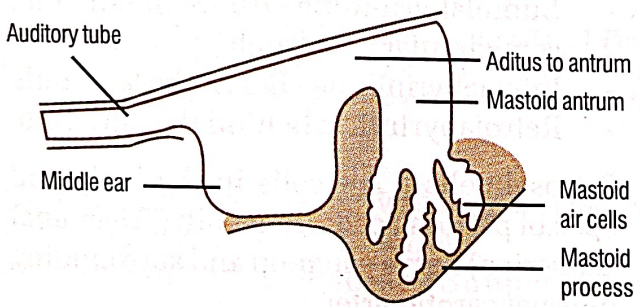
Eustachian tube opening

The aditus to the mastoid antrum

(Aditus ad antrum)

It is an opening in the posterior part of the attic which communicates to the mastoid antrum

The horizontal (lateral) semicircular canal lies on its medial wall where as fossa incudis with short process of incus lies below it. The facial nerve lies below the aditus.



Relationships of the middle ear cavity

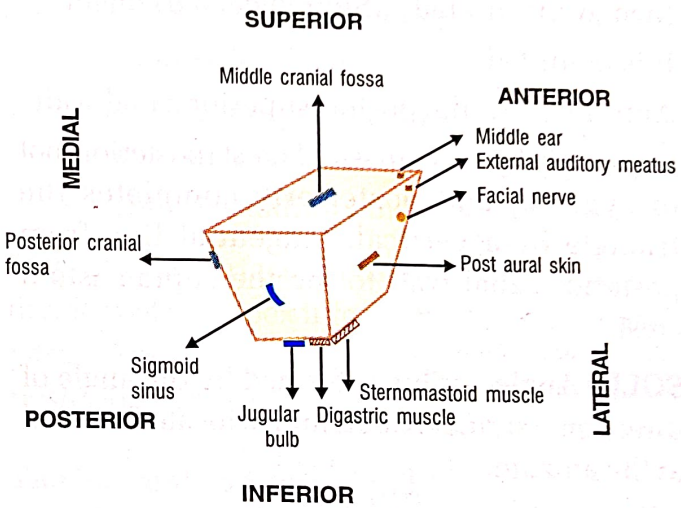
Mastoid antrum

Mastoid antrum is a air containing small, circular space situated in the petrous part of the temporal bone. It is completely developed at birth. Its capacity is about 1ml.
The dimensions of the antrum are

From front to back	14 mm.
Top to bottom	9 mm
Side to side	7 mm

Depth from surface i.e. thickness of lateral wall 12 -15 mm. (At birth - 2 mm)

The antrum is located 1.5 cm behind & below the suprameatal spine of Henle (Suprameatal spine i.e. a small sharp bony projection/ spicule in the postero-superior angle of meatus) in adults. In children's antrum is located superficially. The antrum is always present.



Relationships of the right mastoid antrum

Fossa mastoidea or cribriform area is a small shallow depression 1 cm behind the spine of Henle. In children, it is perforated by many blood vessels.

Relations

Superiorly

- Tegmen tympani (floor of middle cranial fossa)
- Temporal lobe of brain.

Inferior / Floor : Mastoid portion of temporal bone.

Floor

Lateral - Digastric muscle

Medial - Sigmoid sinus

Anterior – Aditus ad antrum in its upper part
(Medial - Ampullae of SSC & LSC)

Lower part - Facial nerve canal

Posterior wall - Sigmoid sinus (then cerebellum) with its bony covering .

Medial - Lateral semicircular canal
Endolymphatic sac lies below
Dura of the Posterior cranial fossa

Lateral - 1.5 cm thick squamous part of Temporal bone

The surface of lateral wall corresponds to Macewen's triangle. (Suprameatal triangle).

It is bounded

Anteriorly by the posterosuperior canal wall

Superiorly by supramastoid crest (posterior root of zygoma) and posteriorly completes the triangle by a vertical, tangential line from posterior canal wall to the the supramastoid crest.

SOLID Angle – This is formed by the angle of three semicircular canals and is located medial to the antrum.

TRAUTMAN'S TRIANGLE – This triangle is formed by the bony labyrinth anteriorly, sigmoid sinus posteriorly and superior petrosal sinus superiorly and is located behind the antrum.

Antrum threshold angle – This triangular area is formed by the horizontal semicircular canal and fossa incudis above, descending part of facial nerve medially and chorda tympani laterally.

Sinodural angle – Angle between the tegmen tympani (i.e. middle fossa dura) and the sigmoid sinus. (Citelli's angle)

Mastoid process

Located behind the tympanic part of the temporal bone. Its development starts at the end of first year of life and completes by puberty. As mastoid process is absent in infants the facial nerve lies immediately behind the tympanic ring, so the chances of facial nerve injury is very high in post aural incisions.

Mastoid air cells

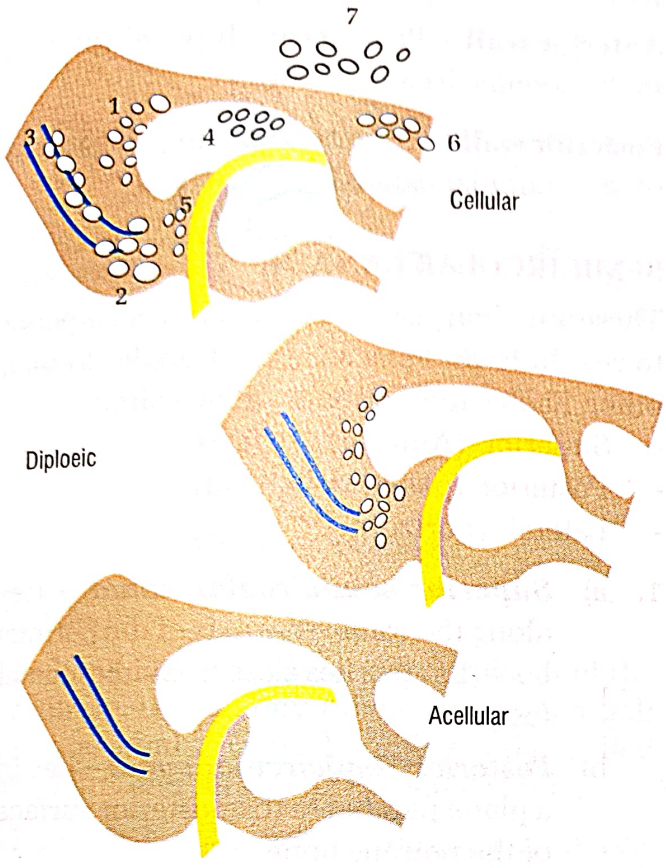
These are intercommunicating air containing sacs/cavities of the mastoid cortex. These cells are lined by flattened nonciliated squamous epithelium.

Normally mastoid bone is cellular. (in 80 % of individual) but in some people (about 20%) there may be Diploic (mixed-Marrow containing interspersed few air cells,persisting from fetal life.) and Sclerotic (Ivory /Acellular - air cells are absent).

Mastoid air cells are named, based on their location as :

1. Periantral cells –
   - Air cells near antrum.
   - Air cells surrounding antrum.
2. Perifacial and Retrofacial cells – Around facial nerve.
3. Perisinus cells – Cells surrounding sigmoid sinus, Sinodural angle.
4. Perilabyrinthine cells –
   - Around the Labyrinth but within the petrosa.
   - Supralabyrinthine - Above the superior semicircular canal arch.
   - Infra labyrinthine - Below the labyrinth.
   - Retrolabyrinth is behind the labyrinth.
5) Petrosal cells – Air cells in the body and apex of petrosa., cells under 5th (Trigeminal /gasserian) nerve ganglion and surrounding Internal carotid artery.
6) Peri tubal cells – Cells around Eustachian tube.
7) Tip cells – of the mastoid
   i) *Superficial cells* - lie superficial to posterior belly of digastric muscle .
   ii) *Deep cells* - lie deep to superficial cells. These cells are separated by digastric ridge. This is the surgical landmark of facial nerve. (anterior to the ridge)
8) Squamous cells – Cells in the squamous part of temporal bone

9) Zygomatic cells – cells extending into the zygoma of temporal bone



The mastoid types and distribution of air cells (1) Peri-antral cells. (2) Tip cells. (3) Perisinus cells. (4) Perilabyrinthine cells. (5) Retrofacial cells (6) Zygomatic cells. (7) Petrosal cells.

These cells when infected will form an abscess and need surgical intervention.

- Post-auricular abscess (Subperiosteal abscess)
- Bezold abscess
- Luc's abscess
- Citelli's abscess
- Zygomatic abscess

Petrous apex

This is a conical shaped, medial part of temporal bone. It has a base and three sides.

Base is by

- Canal for tensor tympani (front to back)
- Carotid artery
- Cochlea (Vestibule ,semicircular canals)

Posterior surface - Bony wall of posterior cranial fossa, which extends from back to front by Endolymphatic sac

- Site of origin of petroclinoid ligament.
- The lower limit of this surface is occipital bone suture line.

Superior surface - is the floor of middle cranial fossa (extends from line of the superior semicircular canals to the foramen lacerum)

- Impression for the trigeminal ganglion.

Structures passing through petrous bone

- Carotid artery.
- Internal auditory meatus.

At posterior surface internal auditory meatus divides petrous apex into anterior and posterior parts. Anterior part contains 10% where as posterior part contains 30% of air cells.

The internal auditory canal

This is a 1 cm long, dura lined short canal. This passes into the petrous bone in a lateral direction from the cerebello-pontine angle.

The Fundus is the outer lateral end, closed by the bone plate through which blood vessels and nerves passes from cranial cavity. This transmits facial nerve, auditory nerve (cochlear and vestibular nerves) and internal auditory artery and vein.

The Crista falciformis is a transverse crest of bony plate on the inner medial surface. This separates fundus from the middle ear and inner ear and divides it into upper and lower regions.

1. ***Upper region*** – This region is divided into anterior part and posterior part by a small vertical ridge, i.e. Bill's bar.

   Anteriorly - Facial nerve
   Posteriorly - Superior vestibular nerve.
   (supplies - superior and lateral semicircular canals, utricle and part of saccule)

1) ***Lower region*** –

Anteriorly – Cochlear nerve (Cochlear area - spiral of small foramina and central canal) i.e. Tractus spiralis foraminosus

Posteriorly – Inferior vestibular nerve (supply - saccule)

Below and behind the inferior vestibular nerve - Singular nerve comes through the foramen singulare and passes through the petrous bone near round window and supply posterior semicircular canal (ampulla)

INNER EAR

This is located in the petrous part of the temporal bone and is an organ of hearing and balance. It consists of bony and membranous parts. The Membranous labyrinth is filled with endolymph and covered by the bony labyrinth. The space between the bony and membranous labyrinth is filled with perilymph.

1) Bony Labyrinth - houses perilymph fluid

This part consists of
- Vestibule
- Cochlea
- Semicircular canals

Vestibule – It is a small central chamber placed between the medial wall of the middle ear and lateral end of the internal auditory canal.

Lateral wall – It is separated from the middle ear by the footplate of the stapes. (Oval window)

Medial wall – Two recesses are present.

1) **Spherical recess** (contains saccule, which carry the Inferior vestibular nerve fibres)
   - Vestibular crest is an ridge located behind this recess
   - Cochlear recess is a area between the divided lower end of crest which takes the Cochlear nerve fibres to the cochlea

2) **Elliptical recess** – Is placed below and behind the vestibular crest and contains macula of utricle .

Below this recess, an aperture of aqueduct of vestibule (a small canal) is located through which endolymphatic duct passes.

Anterior wall – Presents an elliptical aperture in the cochlea (scala vestibuli)

Posterior wall – Presents 5 openings (apertures of semicircular canals)

SEMICIRCULAR CANALS

These are 3 bony canals, located posterosuperior to vestibule and placed at right angles to each other. Each canal forms 2/3rd of a circle.
- Superior (Anterior vertical)
- Posterior (posterior vertical)
- Lateral (horizontal)

1. a) ***Superior semicircular canal*** – lies along the transverse axis of the petrous bone. Its apex lies close to middle cranial fossa.

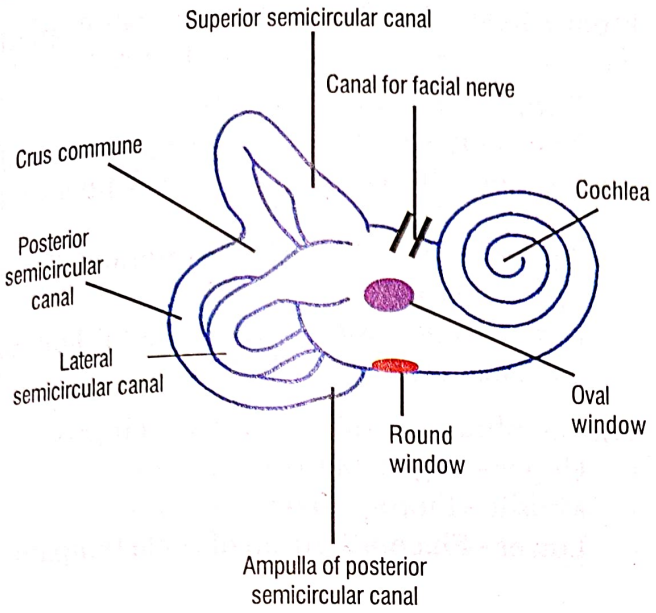
   b) ***Posterior Semicircular canal*** – lies in a plane parallel to the posterior surface of the petrous bone.

   c) ***Lateral semicircular canal*** – lies between the superior and posterior canals. It forms a bulge on the medial wall of the antrum. This slopes downwards and backwards in standing position.

Each canal has ampullated and nonampullated ends. Ampulla is an dilated end of canal which opens independently into the vestibule (contains Vestibular sensory epithelium).

The ampulla of superior and lateral canals lie next to each other anterolaterally, while posterior canal ampulla is located inferiorly. (near the floor of vestibule).

Nonampullated end – The superior and posterior canals nonampullated ends unite to form the "Crus commune" a common channel. This opens in the posterior part of vestibule.



The Bony labyrinth

Cochlea

The bony Cochlea – Is situated in front of the vestibule. It is a bony tube of two and a half turns around a central conical pillar called modiolus and it resembles a snail shell.

The modiolus arises from the cochlear nerve, part of the fundus of the internal auditory canal. It is directed forwards and laterally and ends at the apex.

The modiolus contains numerous small canals, which enter the bony spiral lamina.

Bipolar ganglion cells are located at this place

It has a wide base and narrow apex.

1) Base – thick and directed towards the internal acoustic canal, through which nerves and vessels pass to the cochlea.
2) Apex – tapering part of broad base which is directed towards antero-superior part of medial wall of tympanic cavity.

The bony bulge in the medial wall of the middle ear is called as Promontary which is the basal turn of cochlea.

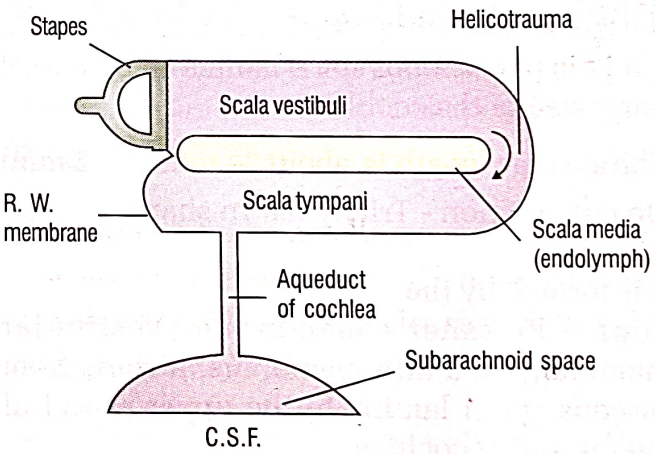
The osseous / bony spiral lamina is a spirally winding, thin shelf bone around modiolus in the lumen of cochlea. ("like the thread of a screw").

The bony spiral lamina divides the cochlea into compartments by the membranous spiral lamina (extends from the bony spiral lamina to the outer wall of cochlea).

- Scala Vestibuli (Upper part)
- Scala Tympani (Lower part) (blind ended tube)
- Scala Media

The scala vestibuli and tympani lie above and below the scala media respectively and contain perilymph. They communicate with each other at the helicotroma which is the apex of the cochlea. Functionally the scala vestibule communicates with the middle ear via the oval window (covered with stapes footplate) and the scala tympani via the round window. (covered with round window / secondary tympanic membrane).

Cochlear aqueduct arises from its basal end and connects it to subarachnoid space.



Perilymphatic system
(CSF passes into scala tympani through Aqueduct of cochlea).

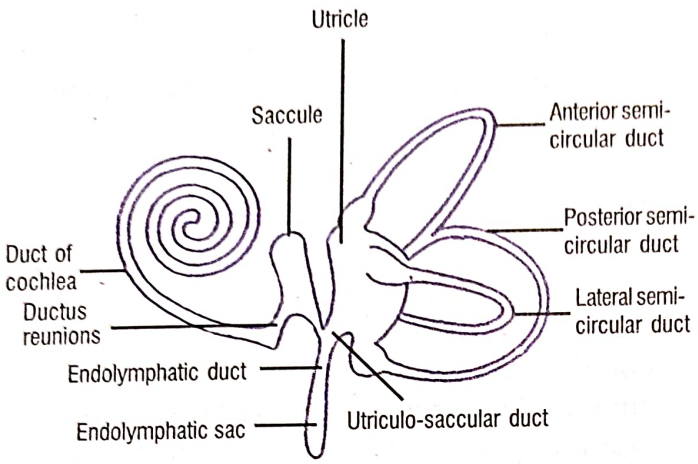
2) Membranous Labyrinth

It is a series of communicating sacs and ducts derived from ectoderm and filled with endolymph. It contains - Cochlear duct.

Membranous / Vestibular labyrinth

- Utricle and Saccule
- 3 Semicircular ducts
- Endolymphatic duct System

(The membranous labyrinth contains endolymphatic fluid which is similar to that of the intracellular fluids).



The Membranous labyrinth (as seen from the lateral side)

I) **Cochlear duct** (Scala Media / Membranous cochlea) / Ductus Cochlearis

This is a coiled membranous duct/tube situated between the scala vestibuli and the scala tympani (the osseous spiral lamina and external bony wall of the cochlea)

The average length is about 34 mm (+/- 2 mm)

On cut section - Triangular in shape

It is formed by the

Roof – Reissner's membrane (Vestibular membrane) is a thin membrane. It starts from osseous spiral lamina to the upper aspect of lateral wall of cochlea .

It consists of special squamous epithelial cells.

Types of cells –

1) Polygonal, flat cells with short microvilli and distributed throughout the membrane but more at spiral limbus area.
2) Rounded cell's arranged in bands, strands, whorls, or clusters.

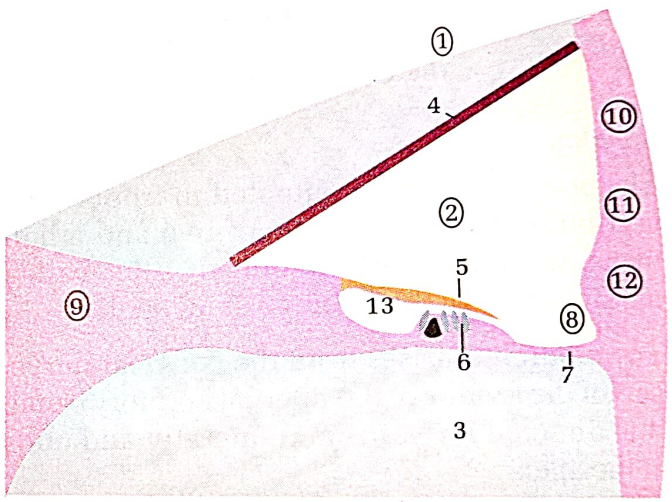
Both these cells are separated by thin membrane. They cells are joined by the tight junctions. These mainly (help to) maintain the usual ionic constitutes of endolymph.

Floor / Base - Basilar membrane (bony spiral lamina and membranous spiral lamina)

- Supports the organ of corti.
- The bony spiral lamina is the inner part of the floor. It gives rise to two (Upper and Lower) ridges.
- Upper ridge - Tectorial membrane (from Spiral limbus)
- Lower ridge - Membranous spiral lamina and Acoustic nerve fibres.

The membranous spiral lamina - 3 layers

- Upper - Organ of Corti
- Middle - Fibrous layer
- Lower - Flat epithelium of scala tympani .



The Cochlear duct. (1) Scala vestibuli 2) Scala media 3) Scala tympani (4) Reissner's membrane (5) Tectorial membrane (6) Organ of Corti (7) Basilar membrane (8) Outer sulcus (9) Spiral lamina (10) Stria vascularis (11) Transitional zone (12) Spiral prominence (13) Inner sulcus.

Organ of Corti

Consists of a series of epithelial structures arranged along the inner edge of the basilar membrane.

A tunnel consisting of two rows of rods of Corti form a triangle with the basilar membrane which divides the organ into an inner and outer portion. On the inner side of the inner rod lies a single row of hair cells, each of which contain stereocillia (70-100) arranged in rows with their apices directed away from the modiolus. The

tectorial membrane overhangs the organ of corti and the stria vascularis lines the outer side of the scala media. The terminal fibres of the cochlear division of the VIII nerve are in contact with the hair cells.

The lateral wall of cochlear duct : has three different zones.

Above - Stria vascularis
Middle - Transitional zone
Below - Spiral prominence

1) The marginal cells – This has a layer of microvilli and is tightly bound with surrounding cells. It has complex foldings of basal membrane. The cell's contains mitochondria, Golgi apparatus and endoplasmic reticulum. These cell's are directed towards the endolymph.
2) Intermediate cells - These cells present in are between marginal and basal cell's.
3) Basal cell layer has rich capillaries.

This active stria maintains the ionic composition and electrical potential of the endolymph.

II) Saccule and Utricle

The saccule is a vestibular sac (globular collection of sensory epithelium) located anterior to the utricle in the scala vestibuli, opposite to the stapes footplate.

The saccule is anteriorly connected to the cochlea by a " Ductus reunions" (short narrow tube).

Posteriorly saccule is connected to endolymphtic duct and makes contact with utricle.

Utricle is an irregular oblong shaped large sac, located in the postero-superior part of vestibular labyrinth. (Above and behind) It receives 5 openings of the semicircular canals.

Both of these contains macula, a comma shaped specialised epithelium which responds to linear acceleration and deceleration. The macula of utricle is located at a right angle to saccular maccule.

Utriculosaccular duct connects utricle with saccule.

In Meniere's disease the distended saccule lies against the stapes footplate and can be surgically decompressed by perforating the footplate.

III) Semicircular ducts

These correspond to three bony canals. They open in the utricle. The ampullated end of each duct contains a thickened ridge of neuroepithelium called 'Crista ampullaris'. The diameter of each duct is about 0.2 mm.

Vestibular sensory epithelium

The ampullae and maculae of both contain type I and type II cells.

Type I cells are flask shaped (Round base with short neck). Body is surrounded by the goblet shaped nerve terminals i.e. Chalice. The cuticular plate is the thick upper part of the cell.

Type II cells are cylindrical in shape with button like nerve terminals.

IV) Endolymphatic Duct

In Meniere's disease the distended sac is decompressed distally and increased pressure can be reduced by shunt operation.

The Utriculoendolymphatic valve of Bast :

This separates the utricle and dilated proximal part of the endolymphatic duct.

Blood supply of Labyrinth

The Labyrinthine (Internal auditory) artery- branch of anterior inferior cerebellar artery or sometimes directly from Basilar artery. This divides into an anterior vestibular and a common cochlear artery the common cochlear artery further divides into the cochlear and vestibulocochlear artery.

The anterior vestibular artery supplies to utricle and semicircular ducts.

The vestibulocochlear artery further divides into terminal vestibular and cochlear branches at modiolus. The vestibular artery supplies to saccule, semicircular canals and basal end of cochlea and then it anastomosis with cochlear artery. The terminal branches of these arteries supply to osseus spiral lamina and ganglion, limbus and the spiral ligament. (Vascular supply is described by Axelsson).

ORGAN OF CORTI

Venous drainage :

1) The Internal auditory vein
2) Vein of cochlear duct
3) Vein of Vestibular Aqueduct

Nerve Supply

1) Vestibular nerve (Formed by the ampullary, utricular and saccular nerves)
2) Vestibulocochlear nerve (Formed by the vestibular and cochlear nerve)

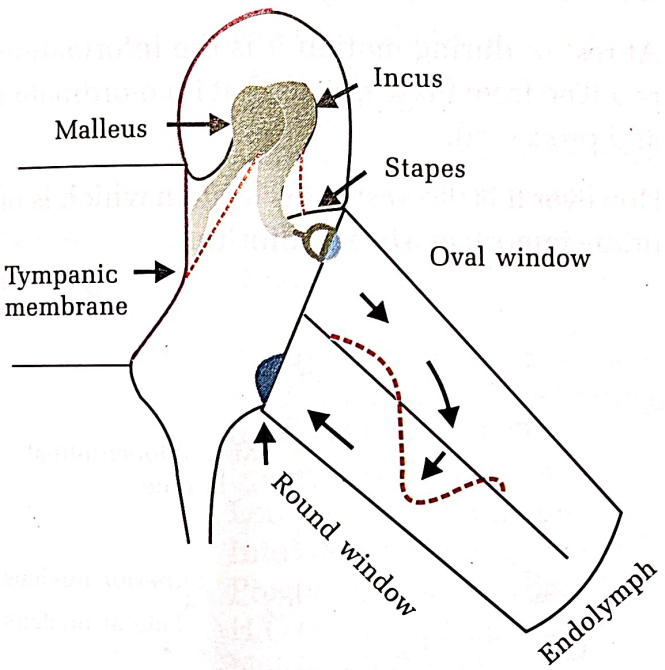
CHAPTER 2

Physiology of the Ear

AUDITORY FUNCTION

This can be divided into two parts

1. ***Sound conducting apparatus*** – External canal, tympanic membrane and ossicular chain and
2. ***Sensorineural apparatus*** – Organ of corti, auditory division of the nerve and its central connections.

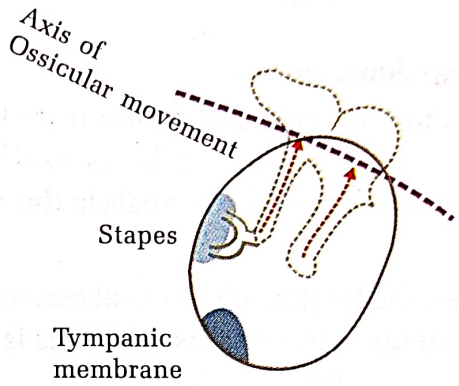


Mechanics of Hearing

External ear function :

The pinna acts as a sound collector, intercepting sound energy and deflecting it into the auditory canal, which by its tortuous shape offers protection against physical violence and entry of foreign material. The auditory canal transmits the sound waves to the tympanic membrane. The tympanic membrane through ossicles transfer the sound waves to the oval window.

Middle ear function :

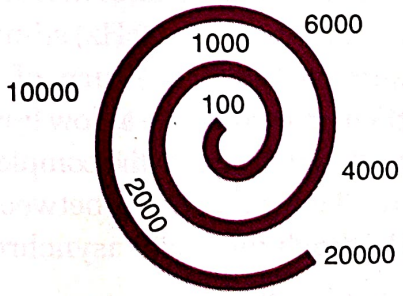


Transformer action of the middle ear

i ***Hydraulic ratio :*** The sound energy exerted on the whole tympanic membrane gets transmitted by an intact and mobile ossicular chain on the oval window. So the accentuation of sound achieved is the ratio between the area of the mobile tympanic membrane to that of the footplate of the stapes i.e. 17 times.

ii. ***Ossicular leverage :*** A mechanical advantage is obtained due to the lever action caused between the handle of malleus to the long process of the incus. The leverage ratio being 1.3:1.

The advantage obtained due to the middle ear transformer mechanisms is 17 x 1.3 = 22 times



Localisation of frequency in the cochlea

The middle ear muscles protect the inner ear from loud sounds.

The sound is also transmitted by the bone conduction. It is brought by the vibration of the skull (including ossicles and labyrinth, mandible) which causes vibration of inner ear fluid.

Inner ear function :

This includes transfer of sound from the oval window to the hair cells as well as its conversion into action potentials in the auditory nerve.

However, the final mechanism of conversion of sound energy into nervous impulses is not yet known.

Theories of Hearing

The cochlear pitch discrimination is explained by the following theories.

1) **Helmholtz's theory (Place theory)** – This theory states that "each pitch vibrates a particular part of the basilar membrane ".

2) **Rutherford's "Telephone" theory** – The uniform vibration of all parts of basilar membrane occurs in response to the sound stimulus and amplitude represents/reflects intensity of sound. The rate of firing of the auditory nerve fibres determine the sound frequency.

3) **Wever's Volley theory (of hearing)** – This theory is a combination of Helmholtz's and Rutherford's theory. It states that the higher frequencies (above 5000 Hz) stimulate the hair cells in the basal turn of cochlea (i.e.particular area) where as low frequencies (below 400 Hz) stimulate the complete organ of Corti. The frequencies between 400 - 5000 Hz reach the CNS asynchronously.

4) **Bekesy's traveling wave theory** – A wave like vibration of the basilar membrane occurs at the oval window in response to sound .The vibration starts from the basal turn of cochlea and reach to the apex of cochlea. It increases in amplitude as it moves till it reaches a maximum and it vanishes. The point of maximum amplitude determines the frequency of sound.

Final perception of sound occurs in the cerebral cortex.

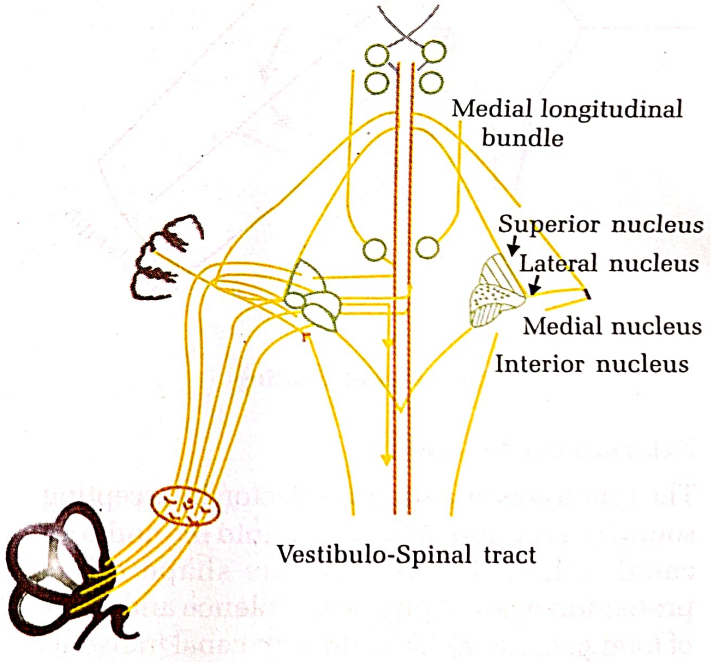
VESTIBULAR FUNCTION

Sensory input necessary for the maintenance of equilibrium is obtained from

i. Visual
ii. Somatosensory and
iii. Vestibular organs

At rest or during motion it is the information received from these organs that is co-ordinated and processed.

However it is the vestibular system which is of prime importance of equillibrium.



Central connections of Vestibular nerve

CHAPTER 3

Examination of the Ear

HISTORY

Complaints –

Ear discharge: Duration, episodic / continuous
Onset - Sudden / insidious
unilateral / bilateral, mucoid / purulent / mucopurulent / blood stained, Colour-yellow / red/ greenish / clear, colourless
scanty / moderate / profuse,-
Amount odourless / foul smelling.
With pain
decrease in hearing
respiratory tract infection
effect with treatment
ear drops coming into the throat (eustachian tube patency)

Decrease in hearing : Unilateral/Bilateral, Duration,
Onset-sudden / insidious,
Congenital / Acquired
Progressive / mild / moderate / severe / constant / fluctuating,
Increased with URTI
Speech discrimination
Loudness discrimination
Intolerence for loud sound
Pregnancy related / familial
H/O Ototoxic drugs
Noise induced trauma affecting routine work
Use of hearing Aids.
H/O Viral exenthematous fever

Earache : Unilateral / Bilateral, Duration
Onset - sudden / insidious
Dull / burning / throbbing / pricking
Localization
Intermittent / Continuous
Aggravating / Relieving factors
Disturbs sleep with respiratory tract infection, relief following ear discharge.

Giddiness : Occassional (Episodic) / continuous description of attacks, frequency of attacks
Duration
Duration of each attack
Onset - sudden / insidious
Rotational / Unsteadiness
warning symptoms, if any-
Associated with nausea & Vomiting
with position of head
Postural / positional variation
imbalance while walking
Aggravating / Relieving factors.

Tinnitus : Unilateral / bilateral
Duration
continuous / intermittent / pulsatile
Onset : sudden / Insidious
Progressive / Nonprogressive
High pitched / Low pitched
Character : roaring / humming / clicking / hissing
Disturbs sleep
with ear discharge
with fullness in the ear
H/O use of Ototoxic drugs

On Enquiry History of : Cold / cough / fever / allergy / trauma (etiology) headache / vomiting / convulsion / postaural swelling / facial asymmetry / visual disturbance (for complications) diabetes / hypertension / tuberculosis (for fitness to undergo surgery)
Operation in the past (for prognosis)
H/O Syphilis / use of Ototoxic drugs
Family / parental h/o decreased hearing i.e. Otosclerosis

General Examination :

LOCAL Examination :

A. **EAR :** right / Left

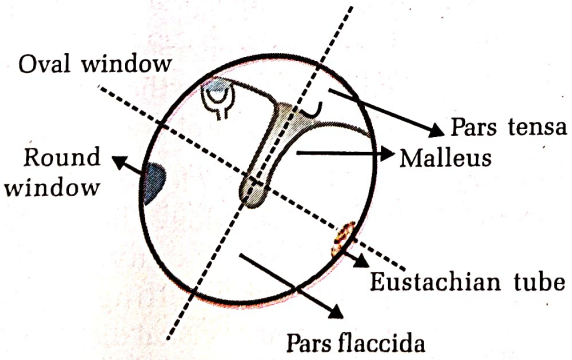
i) ***Preauricular area*** : Seen for sinus, fistula, tag, lymph node or parotid swelling.

ii) ***Post-aural area :*** Seen for scar of previous surgery or a swelling indicating a mastoid abscess.

iii) ***Pinna :*** If malformed would indicate an abnormal ossicular chain also (as both develop from the 1st and 2nd branchial arches). Pinna may be inflamed, traumatized or affected by the neoplasm.

iv) ***External auditory canal :*** To rule out an otitis externa, as it is a contraindication for surgery.
Swelling of canal wall (Furunculosis), wax,
Sagging of posterosuperoir wall (Mastoiditis)
Canal may be red, congested, or granulated.
May show polyp or polypoidal mass (Ca).

iv. Tympanic membrane



Right Tympanic membrane

The various structures seen are – Handle of malleus, cone of light, pars tensa and the pars flaccida.

A perforation in the pars tensa may be either :

Presence and position of perforation and retraction pockets :

a) ***Traumatic*** – Where a slit like perforation in seen with irregular margins

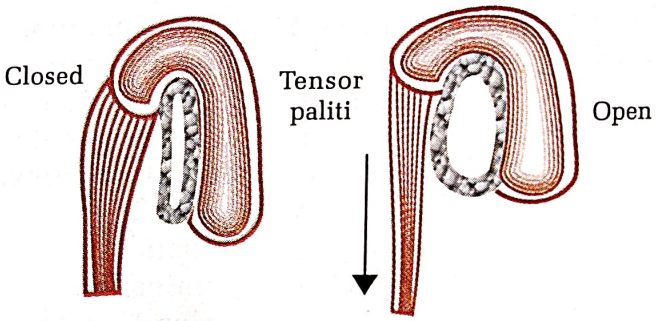
b) ***Marginal*** – As the perforation lies at one of the edges, thus the tympanic membrane does not surround the full perforation.

c) ***Central*** – Which is surrounded by remnants of tympanic membrane all around and occupies all 4 quadrants of the pars tensa. Depending upon its size, the perforation is called a small, moderate or a large sized central perforation.

d) ***Subtotal*** – Perforation is surrounded by the tympanic annulus due to absence of normal tympanic membrane.

e) ***Total*** – Perforation occurring due to loss of tympanic membrane as well as the annulus.

Through a perforation one can see the middle ear mucosa, type of discharge, incudostapedial joint, round window and the eustachian tube opening.



Eustachian tube opening

v. ***Valsalva's manoeuvre*** – To test for eustachian tube patency (a pre-requisite for a tympanoplasty).

vi. ***Mastoid tenderness*** – Elicited at the area overlying the antrum.

vii. ***Fistula test*** – To check for an abnormal communication between the middle ear and the inner ear caused by a cholesteatoma.

viii. ***Tuning fork tests*** – To determine the side (right/left), type (conductive/sensorineural) and the degree (mild/moderate/severe) of hearing loss.

ix. ***Nystagmus*** – Is tested for as may occur due to labyrinthitis following otitis media.

x. ***Facial paralysis*** – In seen for as it is one of the commonest complications of otitis media.

xi. ***Cerebellar and Meningeal signs*** – To rule out other intracranial complications.

B. Nose

C. Throat and

D. Systemic examination

Diagnosis :

i. Unilateral / Bilateral
ii. Acute / Chronic
iii. Suppurative / Non-Suppurative Otitis media
iv. Mild / Moderate / Severe
v. Conductive / Sensorineural / Mixed Hearing loss
vi. Associated Nose / Throat Pathology
vii. In an Active / Inactive State, with
viii. Intracranial complications (if any).

Investigations :

1. ***Ear Microscopy*** –
   i. Confirmation of ear findings
   ii. Detection of hidden cholesteatoma
   iii. Ossicular chain assesment (if possible)
   iv. Collection of ear discharge for smear, culture and antibiotic sensitivity (SCABS).

2. ***Hematocrit*** – for assesing the general condition of the patient before surgery.

3. ***Puretone Audiometry*** –
   i. To know the exact hearing loss.
   ii. For documentation of hearing, required to compare the operative results.
   iii. For medico-legal purposes.

4. ***Radiology :***
   a) ***X-ray Mastoid. (Schuller's view)***
   i. Destruction of the mastoid bone is observed, which is seen as a cavity.
   ii. To know the limits of the mastoid air cells before a mastoidectomy.

   b) ***X-ray paranasal sinuses (Water's and Caldwell's view).***
   To rule out a focus of infection in the paranasal sinuses.

OPERATIONS

Myringoplasty
Tympanoplasty
Mastoidectomy

EUSTACHIAN TUBE PATENCY

Found out by the following :

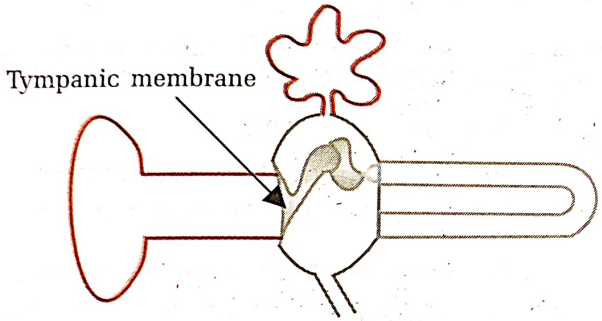
1. ***History*** – Of ear drops coming into the throat, indicate a patent eustachian tube.

2. ***Valsalva's manoeuvre*** – Is forced expiration on a closed glottis. The patient is asked to keep his mouth closed, pinch nostrils and then to breath out. With this action an outward movement of the tympanic membrane is seen. However, the drawbacks of this procedure are
   i. It can be only performed during the expiratory phase of respiration.
   ii. The pressure built up during the manoeuvre is very high and therefore could lead to an inner ear damage.
   iii. A positive intrathoracic pressure which builds up causes peripheral pooling of blood and resultant hypoxia.
   iv. The patient has to be taught the procedure.

3. ***Politzerisation*** – Is done by fitting the nozzle of the Politzer's bag into one nostril of the patient while the other nostril is pinched. The patient is asked to perform the swallowing movement of the soft palate with closure of the nasopharynx, thus pushing air into the middle ear via a patent eustachian tube.

4. ***Toynebee's manoeuvre*** – Is swallowing mechanism along with the nostrils being closed. This produces a rise of pressure in the nasopharynx followed by a negative pressure, leading to tympanic membrane movement if the eustachian tube is patent.

5. ***Frenzel's manoeuvre*** – Is voluntarily closing the glottis, mouth and nostrils, while simultaneously contracting the muscles of the floor of the mouth. This technique has to taught and learned. The advantage being, it can be performed in any phase of respiration and is independant of the intrathoracic pressure.

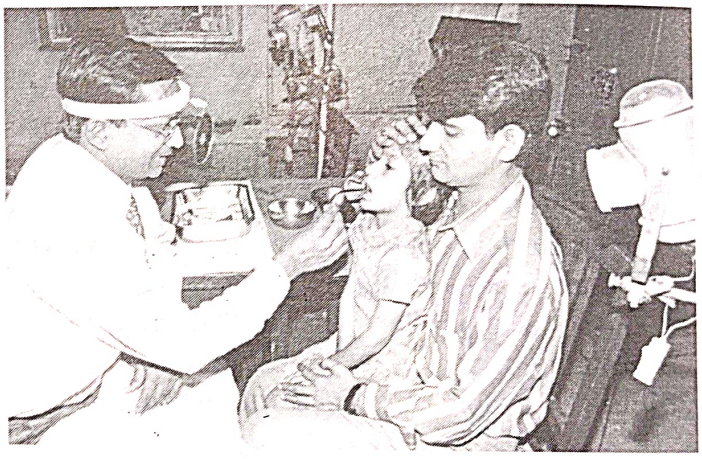


Eustachian tube

6. ***Eustachian tube catheterisation.***
In this test eustachian catheter is passed along the floor of nose, once it reached nasopharynx, tip is rotated medially to touch posterior part of septum. Then it turned laterally and it enters into the eustachian tube opening.

7. ***Impedance Audiometry*** – Showing a change of pressure in the middle ear.

8. ***Radiology*** – Findings show the eustachian tube boundaries after a radio-opaque dye is injected into the middle ear.



Method of holding a child for ENT examination

CHAPTER 4

Auditory Function Tests

Types :

I. ***Voice tests***

II. ***Tuning fork tests***
   a. Rinne's test
   b. Weber's test
   c. Absolute Bone Conduction

III. ***Audiometry***
   a. Puretone Audiometry
   b. Speech Audiometry.
   c. Free field Audiometry.
   d. Bekesy Audiometry.
   e. Impedance Audiometry.
   f. Evoked Response Audiometry. (ERA).

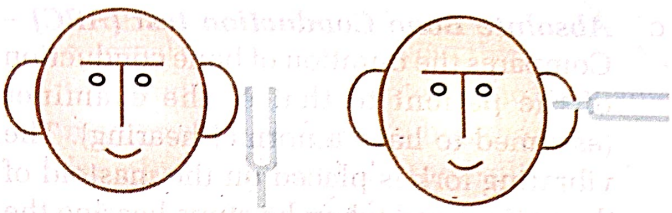
I. VOICE TESTS

In the past, voice tests were used for assesment of the patients hearing capacity by noting the distance at which the patient can hear the examiner's voice. Such a method is inexact as variations in intensity of the examiner's voice may occur and also the test must be performed in a sound proof room.

II. TUNING FORK TESTS

a. ***Rinne's Test :*** Is based on comparing the duration of bone conduction with that of air conduction of one ear. The tuning fork, after being struck is placed on the mastoid process and when the patient stops hearing the sound, the fork is quickly transferred to a position 2 cm lateral to the external meatus. If the patient continues to hear the fork , the Rinne's test is positive and if the patient hears the fork better by bone conduction (BC) than by air conduction (AC), the test is negative.

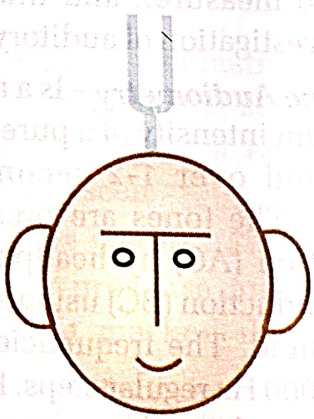
RESULT	INTERPRETATION	DIAGNOSIS
+	AC>BC	Normal or Sensorineural loss
=	AC=BC	Mild Conductive loss
-	BC>AC	Conductive loss



Air conduction Bone conduction

A false negative result may occur whenever a patient has severe sensorineural hearing loss in the test ear with a good hearing in the other ear. Such a patient would not hear the tuning fork by air conduction but when the fork in placed on the mastoid, the sound traverses freely through the skull and is heard by the non-test ear. Since the patient cannot localise the sound, his reporting of the sound is wrongly noted as BC > AC i.e. a conductive hearing loss. However, this would be unearthed by a Weber's test.

b. ***Weber's Test*** – Involves placing the vibrating tuning fork against the midline of the skull, either at the vertex or on the forehead.



Weber's Test

Results :

i. ***Normal*** – the patient hears sound equally in both the ears.

ii. ***In Conductive hearing loss*** – The patient lateralises the sound to the affected ear. In case of deafness in both the ears, sound is lateralised to the worse ear.

iii. ***In Sensorineural hearing loss*** – The sound is lateralised to the better hearing ear.

c. ***Absolute Bone Conduction test (ABC)*** – Compares the duration of bone conduction of the patient to that of the examiner (assumed to have a normal hearing). The vibrating fork is placed on the mastoid of the patient and when he stops hearing the sound, the fork is quickly transferred to the examiner's mastoid. During this test, the external canal is occluded so as to allow only the bone conducted sounds to enter. (drawback of Schwabach's test).

Results :

i. ***Normal*** – Being when the absolute bone conduction (duration of sound) is the same for the patient as well as the examiner.

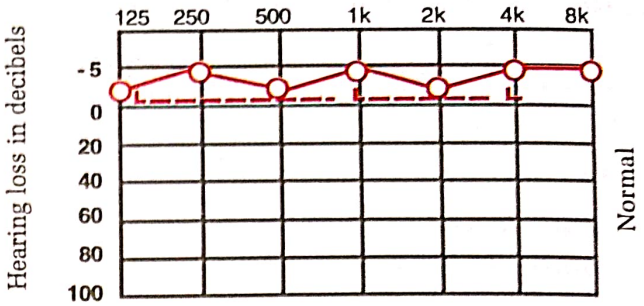
ii. ***In Sensorineural hearing loss*** – The duration of absolute bone conduction of sound is less for the patient than the examiner.

III. AUDIOMETRY

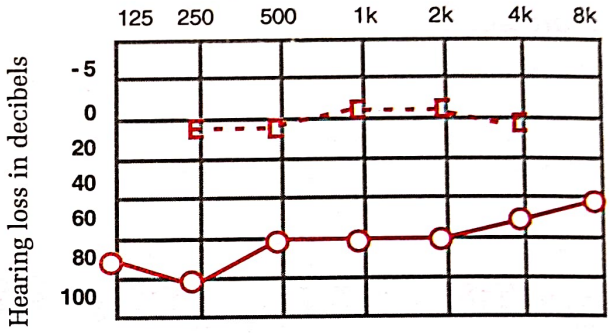
The term is derived from 'audire' (to hear) and 'metrios' (to measure), and this in the most relevant investigation of auditory dysfunction.

a) ***Puretone Audiometry*** – Is a measure of the minimum intensity of a puretone stimulus presented over 1-2 seconds,which is audible. The tones are presented by air conduction (AC) by headphones and for bone conduction (BC) using a vibrator over the mastoid. The frequencies range from 125 to 8000 Hz regular steps. However , this has to be performed in a sound proof room. It is conventional to record the right ear findings with red and left ear with blue, the AC is marked by as a continuous line and BC by an interrupted line.

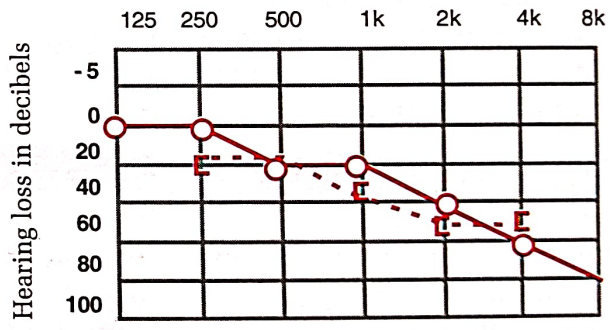
Puretone Audiograms



Normal



Conductive hearing loss



Sensorineural hearing loss

Results :

i. ***Normal hearing*** – Would have a threshold for AC and BC within 20 decibels (db).

ii. ***Sensorineural hearing loss*** – Shows a fall in thresholds for AC and BC.

iii. ***Conductive hearing loss*** – Is diagnosed when the AC is reduced but the BC is normal. The gap between the AC and BC determines the amount of conductive hearing loss and the patient is said to have a mild / moderate / severe conductive hearing loss.

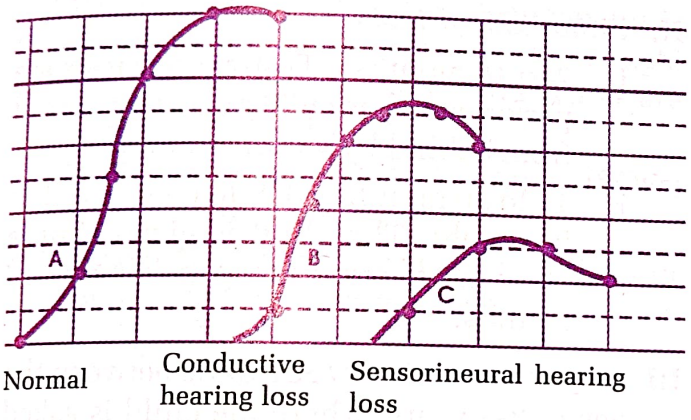
b) ***Speech Audiometry*** – Is a measure of the practical consequences, in terms of ability to communicate with others in a patient having hearing loss.

Results :

i. ***The normal speech curve*** – Has a sigmoid shape and reaches 100% score.

ii. ***A conductive hearing loss*** – Produce a similar curve but at higher intensity of sound.

iii. ***With sensorineural hearing loss*** – The patient fails to reach the 100% mark.



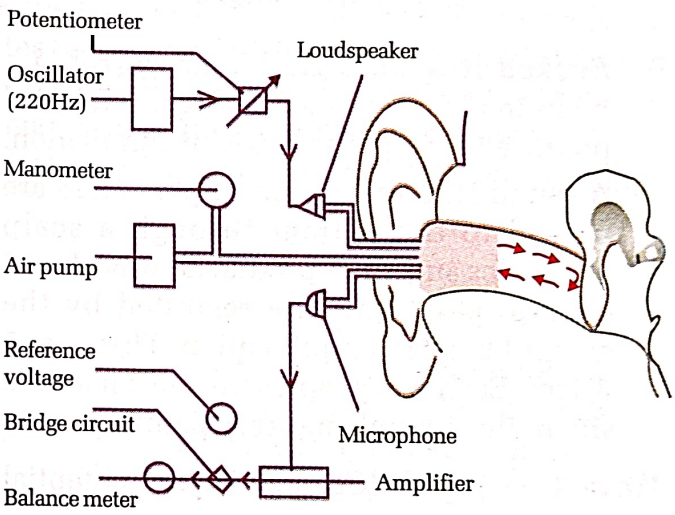
Speech Audiometry

Speech Reception Threshold (SRT) – Of the patient is the minimum intensity of sound at which he can identify 50% of the words correctly. It is measured in decibels.

c) ***Free Field Audiometry*** – Is performed in a small child who would comply to the using of headphones or vibrators. The hearing threshold of the child is obtained by the use of loud speakers in a sound proof room.

d) ***Bekesy Audiometry*** – Is performed to differentiate between a sensory (cochlear) and a neural (retrocochlear) type of hearing loss. This information is not obtained by any of the above forms of Audiometry. Here, the signal frequencies are swept from low to high over a period of about 10 minutes while the intensity of sound is maintained at a just audible level by the patient himself, operating the switch. Thus the difference between the thresholds obtained by the patient for the continuous and intermittent signals diagnose a sensory or a neural hearing loss.

e) ***Impedance Audiometry*** – A low frequency sound (220 Hz) is introduced into the sealed external auditory canal. Now, the impedence to sound would be greater if the tympanic membrane is stiff than when the tympanic membrane is mobile (compliant). The various types of graphs obtained are :



Impedance Audiometry

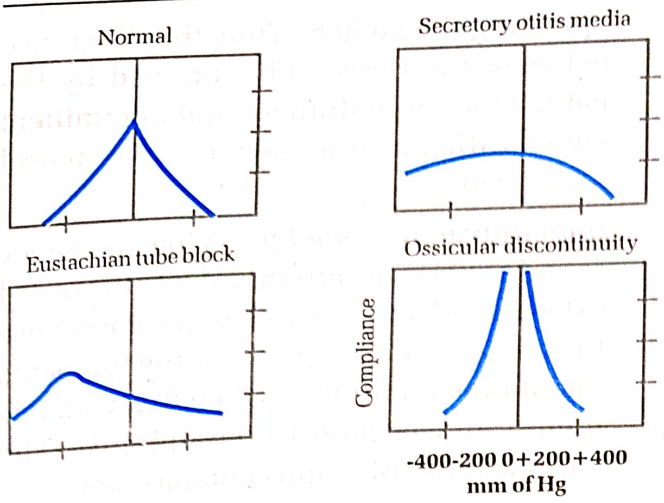
Results :

i. ***Normal :*** When a symmetrical graph with maximal compliance is seen at 0 mm of pressure. Averagely a range 0.6 cc of compliance is seen.

ii. ***Serous otitis media :*** Shows a negative middle ear pressure and a flat curve indicating a marked decrease in the compliance.

iii. ***Ossicular discontinuity :*** Shows a tall and disjoined peak.

iv. ***Otosclerosis :*** Leads to a normal shaped graph but with a decreased compliance, usually less than 0.3 cc.



Types of Tympanogram

f) ***Evoked Response Audiometry (ERA)*** – helps to record the electric signals that are produced in response to sound stimulation.

A sound stimulus i.e. multiple clicks are passed to the patient through a scalp electrodes and thus produced (wave form) electric potentials are recorded by the computer after amplification. There are 5 waves. Each wave represent one anatomic site in the neural auditory pathway.

Wave I – Eighth (Cochlear) nerve potential

Wave II – Cochlear nucleus

Wave III – Olivary complex (Superior)

Wave IV – Lateral leminscus

Wave V – Inferior colliculus

The presence of wave I and absence of waves II to V indicate Retrocochlear pathology. And delayed wave I with normal interpeak latencies in waves I to V indicate conductive or cochlear pathology.

Advantages

i. Can be used in children as well as neonates.

ii. Exact site of lesion can be detected in cases of sensorineural hearing losses.

iii. It is a non-invasive test, so can be repeated any number of times.

iv. The test does not require patient compliance, so useful to detect a malingerer.

HEARING TESTS IN CHILDERN

The earlier an auditory disease is diagnosed, the better are the chances helping a child to overcome the handicap.

I. *Reflex tests*

i. At birth - Moro or Startle reflex

ii. 3 months - Blinking on response to sound.

iii. 5 months - Eyes turn towards sound.

iv. 6 months - Head turns towards sound.

II. *Behavioural tests*

i. 7 to 18 months – Distractions tests are performed . The child may be distracted from behind by various sounds.

ii. 18 to 30 months – Child co-operartion is sought. The child is given simple tasks to perform in response to certain sounds.

III. *Performance tests :* Are done between the age of 2-5 years, where the child is asked verbally to perform tasks.

Pure-tone audiometry

IV. *Subjective tests :*

i. Peep show audiometry : Where a young child is conditioned to press button when a sound is heard.

ii. Conditioned audiometry : The child is taught to build a tower of bricks each time he hears the (test) sound, also referred to as play audiometry.

V. ***Objective tests*** : Are like Impedance Audiometry and Evoked response audiometry. However, these tests may show the integrity of parts of the auditory pathway but do not show whether the child is capable of understanding the sounds.

Otoacoustic emissions

These are acoustic signals produced by the normal cochlea which can be recorded by placing a microphone receiver in the deep meatus.

The recording of cochlear sounds by placing a microphone receiver in the deep meatus is called otoaccoustic emissions. These are thought to be emitted from outer hair cell's. There are two types of emissions.

1) Spontaneous Otoacoustic Emissions (SOEs) Not much diagnostic value.
2) Evoked Otoacoustic Emissions (EOEs or Kemp echoes or Cochlear echo).

In this method cochlear sounds are recorded by computer after sound stimuli.

The evoked response of low frequency sound occurs from apical region where as high frequency sound occurs from basal turn of cochlea. These true sounds are absent when the hearing loss is greater than 30 db. This can be used as a screening test for newborn infants. There absence probably indicates some pathology in the middle ear or cochlea (i.e. integrity of outer hair cells) which needs further investigations (like impedence audiometry, Electrocochleography, and BERA etc). These are easy to perform, quick, fast, non-invasive, sensitive, objective and cochlear informative tests, especially in high risk neonates and infants. However their absence does not necessarily indicate cochlear pathology, (as serous otitis media can affect its detection).

CHAPTER 5

Vestibular Function Tests

I. NYSTAGMUS :

It is a to and fro rhythmic oscillation of the eyes. They are classified as :

1. Vestibular nystagmus
2. Non-Vestibular nystagmus.

Vestibular nystagmus has a characteristic type. The eyes slowly deviate towards the side of paralytic lesion and then quickly jerk back towards their starting point. The direction of nystagmus is named after the direction of the fast component. The degree of nystagmus is as follows -

a) Left nystagmus is of first degree when it occurs on left gaze.
b) Of second degree when it is present on straight ahead gaze.
c) And third degree when present in addition, on the right gaze.

A spontaneous vestibular nystagmus may be due to a peripheral or a central lesion.

II. CALORIC TEST

It essentially consists of obtaining cold and warm caloric responses from each ear, with the temperatures equally above and below body temperature. The patient lies supine with the head inclined to 30°C to bring the lateral Semi-circular canal into the vertical position. Each ear is irrigated with water at 44°C and then 30°C. The duration of nystagmus in noted and results interpreted as :

i. ***Normal*** – Where the duration of nystagmus is almost the same in all 4 tests. Between 60 to 120 seconds.

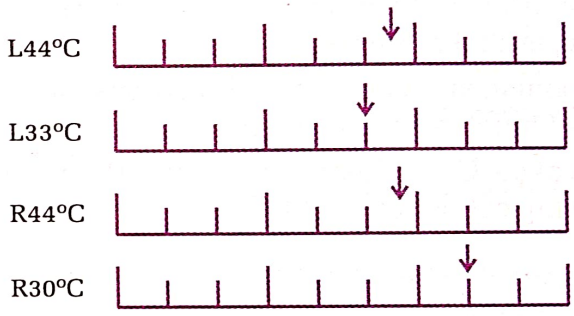
ii. ***Directional preponderance*** – Occurs when the nystagmus shows preponderance to a particular direction, either the left or right.

iii. ***Canal paresis*** – is diagnosed when the duration of nystagmus is shortened on one side, for both 44°C and 30°C caloric tests. This is seen with disease of the semicircular canal.

Contraindications :

A) ***Perforation*** – Would result in spread of infection to the middle ear.

B) ***Acute vertigo*** – First controlled with medications but labyrinthine sedatives should be stopped before performing the test.



Normal Caloric test

Advantages of Caloric test

1. Each labyrinth is tested separately, so the affected side can be found out.
2. A simple test, done in the OPD as does not require any sophisticated equipment.

III. COLD CALORIC TEST

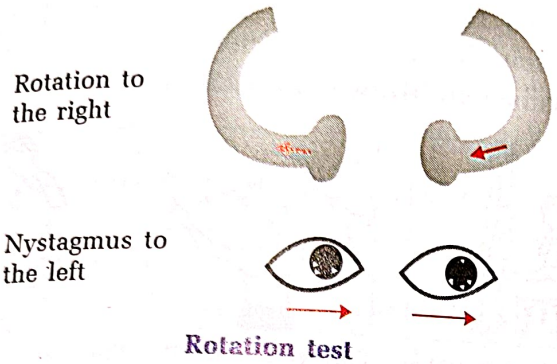
This test is performed when no nystagmus is seen by the above test. Here, the external auditory canal is flushed with a small quantity of ice cold water. Absence of nystagmus even with this test indicates a complete canal palsy.

IV. DUNDA GRANT'S TEST

Also known as the cold caloric test but is to be performed in the presence of a perforation of the tympanic membrane. Here, cold air is supplied by an apparatus which results in nystagmus. The interpretation is the same, as for the above tests.

V. ROTATION TEST

This is performed with the patient seated on a chair which is turned in ten complete circles in a period of 20 seconds. Nystamgus results only if the labyrinths are functioning and the direction of nystagmus is always opposite to that of the rotation.



Rotation test

Disadvantages

1. Expensive equipment is required for this test.
2. Stimulates both the labyrinths, so the side of lesion is not detected.
3. The stimulus is excessively violent, so can even cause damage to the labyrinth.

VI. ELECTRONYSTAGMOGRAPHY

Method of detecting and recording of nystagmus which is induced by caloric, positional, rotational or optokinetic stimulus.

Records the movements of the eye, with the help of electrical potentials that are present between the cornea and the retina.

Advantages

1. Detection of nystagmus, which even may not be visible to the clinican.
2. The nystagmus can be documented for future reference.

VII. FISTULA TEST

Fistula test is performed by alternatingly pushing and releasing the tragus, this causing compression of air in the external auditory canal. The test can also be performed using a Siegle's speculum or an otoscope fitted with a rubber tubing. An abnormal communication (fistula) occurs usually in the bony wall of the lateral semicircular canal resulting from erosion of bone by a cholesteatoma would cause movement of the endolymph with change of pressure in the middle ear. Thus the patient gets vertigo or nystagmus which is observed on performing this test.

Drawbacks :

A. *False Positive results*

i. *In Meniere's* disease due to an abnormally distended saccule being irritated by the slightest movement of the stapes, which is just above it.

ii. *In Syphillis :* it occurs due to the laxity of the annular ligament of the stapes and the osteitis of labyrinth bone.

The false positive result is also known as Hennebert's sign.

B. *False negative results*

No signs of vertigo or nystagmus obtained even in the presence of a fistula, causes

i. ***Cholesteatoma*** – May itself block the fistula.
ii. *A dead or non-functioning* labyrinth.
iii. *Faulty technique of* performing the test. Here, an air seal does not take place and the air pressure build-up does not occur.

The fistula test can also be performed with the Seigle's pneumatic speculum.

VIII. GAIT

Gait is noticeably unsure and wide-based in bilateral vestibular disorders. In cases of unilateral lesions it later becomes normal due to compensatory mechanisms.

IX. RHOMBERG'S TEST

This in useful in cases of unilateral lesions (completely compensated) and bilateral vestibular damage. Here, eye closure results in a dramatic loss of balance, even if proprioceptive sensory function (posterior column) is intact.

Vestibular disorder : The patient sways to the side of the lesion.

Central disorder : The direction of swaying changes on repeated testing.

Hysteria : The patient may fall straight backwards.

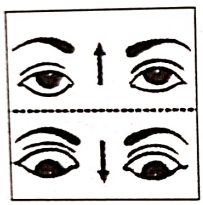
X. POSITIONAL NYSTAGMUS

Positional nystagmus occurs when the patient keeps his head in certain positions. This is corrected by giving the patient head-neck exercises to perform which result in habituation and thus disappearance of vertigo.

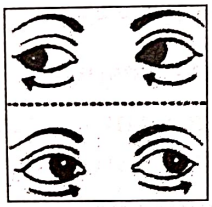
ADAPTATION EXERCISES FOR VERTIGO / DIZZINESS PATIENTS

Exercises in Bed :

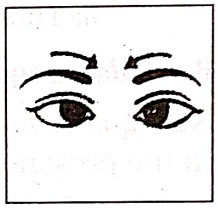
1. Eye Movements



Looking up and then down



Looking alternatively left and right



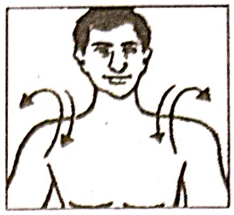
Convergence exercises

2. Head Movements

Bending alternately forward & backward

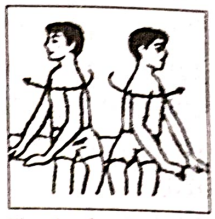
Turning alternately to left and then right

Exercises in Sitting Position :



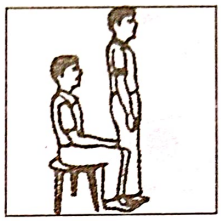
Shrugging & rotating shoulders

Bending forward & picking up objects



Turning head and trunk alternately to the left and right

Exercises in Standing Position :



Changing from sitting to standing, initially with eyes open & then with the eyes shut.

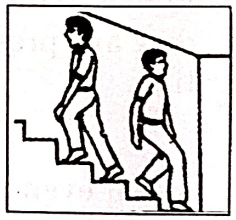
Throwing a small (ping pong) ball in, an arc from hand to hand following it with the eyes

Throwing a small ball from hand to ahnd under the knee

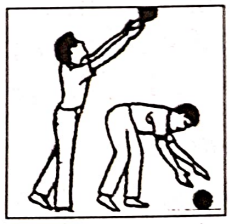
Exercises While Walking :

Throwing and catching the ball while walking

Walking around in the room with eyes open and closed



Walking up and down a flight of stairs



Playing any game involving bending, stretching and aiming with the ball

CHAPTER 6

Diseases of the External Ear

CONGENITAL MALFORMATIONS

Congenital defects of the external ear are associated with defects in the middle ear due to their common embryologic origins.

Types :

1. ***Complete or partial absence of Pinna*** – May occur due to failure of development of the six auricular tubercles.
2. ***Pre-auricular sinus or cyst*** – Occurs due to incomplete fusion of the tubercles.
3. ***Accessory auricles*** – Due to tubercles situated at other sites.
4. ***Atresia of the external auditory canal*** – Due to the defective inward growth and canalisation of the epithelial anlage.
5. ***Abnormalities of shape or size of the auricle*** – e.g. bat ear (protruding ear).

Treatment

1. *Minor auricular defects* – Plastic surgery to improve the appearance.
2. *Major auricular defects* – The use of a prosthesis (artificial pinna) is more effective.
3. *Bat ear* – The cartilage is incised so as to weaken its outwardly projecting spring action and to create a new strong anti-helix.
4. *Deafness* – Is relieved especially in cases of atresia of the external canal.

Pseudocyst of Pinna : It is a cystic swelling in the upper part of pinna. The etiology is not known. It may be due to congenital embryonic dysplasia.

It is treated by high doses of steroids (Prednisolone 1 mgm/ kg body weight in divided doses) for 4 weeks. If not subsided, then Aspiration or incision and drainage done under aspetic conditions and tight pressure bandage should be done.

Keloid of AURICLE

It is a common benign tumor of ear which occurs after ear piercing, trauma or any auricular surgery. Usually occurs at lobule, pinna or helix. These are difficult to remove, as excision itself will cause a larger keloid. The preferred management is intralesional steroid injection.

HEMATOMA OF THE PINNA

It occurs due to rupture of vessels in the perichondrium, which then leads to effusion of blood.

Complications

1. ***Perichondritis*** – If it gets infected.
2. ***Cauliflower ear*** – Occurs due to failure to treat the clot, resulting in a permanent thickening of the pinna.

Treatment

1. Aspiration is done for small clots, while an incision is made to drain large clots.
2. Then, a compression dressing is given around the pinna to prevent recollection of fluids.

Haematoma of pinna / auricle : The trauma to the auricle / pinna causes collection of blood in between the cartilage and perichodrium resulting in swelling of pinna.

It is usually seen in Rugby players and Boxers, ("Boxers ears"). It should be treated by aspiration or incision of swelling (incision should be placed parallel to the helical margin) and tight pressure bandage.

Complication : Cauliflower ear

PERICHONDRITIS

It is the infection of the perichondrium of the auricle.

Most commonly it occurs after injury to the ear, as a complication of haematoma or sometimes after ear surgery. It is usually caused by the Pseudomonas and other mixed organisms. The pinna is red (shiny), uniformly enlarged and tender to touch.

Treatment :

1) A intravenous broad spectrum antiboiotic's
2) Analgesics and anti-inflammatories to reduce pain and inflammation
3) Local application of Glycerine and Ichthammol or Magnesium sulphate or 4% Aluminium acetate.

Complications :

Subperichondrial abscess (collection of pus in between the perichondrium and underneath cartilage. It should be incised only after eliciting fluctuation).

Relapsing polychondritis :

It is an autoimmune inflammatory disease of connective tissue. It may also affect tracheal and laryngeal cartilages. It is treated by high doses of steroids.

KERATOSIS OBTURANS (CANAL WALL CHOLESTEATOMA)

It is the collection of desquamated squamatous epithelium in the inner bony part of external auditory canal, which may erode the bony wall hence referred as cholesteatoma of ear canal. It due to collection of abnormal migrated squamous epithelial cells with wax. It is often associated with bronchiectasis and sinusitis in adolescent patients and those with COPD.

Symptoms :

1. Pain
2. Decreased hearing
3. Ear discharge

Signs :

1) Pearly white mass
2) Conductive deafness

(Its pressure may erode the bony canal wall, so it is referred as choleasteatoma)

Treatment : It is removed under general anaesthesia.

Regular follow up to prevent further occurrence.

FOREIGN BODY

The types of foreign bodies that are found in the external canal may be :

1. Living e.g. maggots or insects.
2. Non-Living e.g. stones, bead, hygroscopic foreign bodies like pea, bean etc.

Symptoms

a. Pain due to a traumatic foreign body.
b. Irritation following swelling up of a hygroscopic foreign body.
c. Noise in case of living insect.
d. Cough (stimulation of the Vagus nerve).

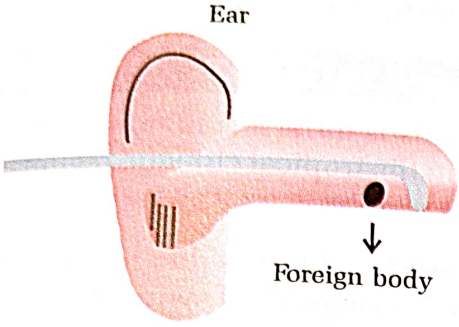
Management

1. ***Living foreign bodies*** – Are killed by borospirit ear drops and then syringed out.
2. ***Non-Living foreign bodies*** – Are removed with a wax hook. Forceps must never be used for removal as they push the foreign body dangerously further in. Impacted hygroscopic foreign bodies may be removed after using glycerin drops, which would shrink the foreign body.
3. ***General Anaesthesia*** – May be required for the above procedure in an unco-operative child or for impacted foreign bodies.

Complications

1. Otitis externa
2. Damage to the tympanic membrane or ossicles by instrumentation.

3. The foreign body might get lodged into the middle ear, which then has to be removed via a postaural incision.



Foreign body removal

OTITIS EXTERNA

Primarily occurs in the external auditory canal or may be a manifestation of a generalised skin condition.

Clinically classified as :

a) Furunculosis (circumscribed otitis externa)
b) Diffuse otitis externa

A more useful classification would be

I. *Infective*
   a. Bacterial
   b. Viral
   c. Fungal

II. *Reactive*
   a. Eczema
   b. Seborrheic dermatitis
   c. Neurodermatitis

I. *INFECTIVE OTITIS EXTERNA*

A) *BACTERIAL INFECTION*

Predisposing factors

1. ***Genetic*** – narrow canal, inherant tendency to eczema, excessive wax formation
2. ***Traumatic*** – match sticks, hair pin etc.
3. ***Environmental*** – heat, humidity and swimming.

Symptoms

1. Pain is severe and aggravated by movement of the jaw.
2. Ear discharge is purulent and blood stained.
3. Deafness if the lumen is blocked due to a furuncle, debris or edema.
4. Trismus following limitation of movement of temporo mandibular joint.
5. Itching.

Signs

1. The external canal appears congested, edematous and stenosed.
2. Pain occurs on movement of the pinna.
3. Discharge may be present.
4. Trismus

This conditions may mimic mastoiditis, the differentiating features being.

	Otitis externa	Mastoiditis
i. History	Trauma / instrumentation water entering the ear	Symptoms of discharge /deafness common
ii. Respiratory tract infection	Absent	Common
iii. Pinna position	Normal	Displaced forwards, Downwards and outwards
iv. Mastoid tenderness	Absent	Present
v. Pinna movement	Painful	No pain
vi. Postaural groove	Normal	Obliterated
vii. External canal	Stenosed and edematous	Normal
viii. Tympanic membrane	Normal	Congested or perforated
ix. Postaural lymphnode	Enlarged	Not palpable
x. X-ray mastoids	Normal	Haziness or destruction of Air cells seen.

Treatment

1. ***Glycerin Icthammol (10%)*** – The pack is placed into external canal. Glycerin reduces the edema by its hygroscopic action and icthammol acts as an antiseptic.
2. ***Heat application*** – Is helpful in reducing the pain and inflammation.
3. ***Antibiotics*** – Are given only in cases of marked edema or when local lymph nodes are palpable.
4. ***Analgesics*** – To reduce pain.
5. ***Incision*** and drainage – Under aseptic conditions in furunculosis, in cases of failure of relief from pain.
6. ***Diabetes*** – Is ruled out in patients having recurrent attacks of otitis externa.

B) VIRAL INFECTION

1. ***Herpes simplex*** – Occurs most commonly on the lips, however may be occasionally found involving the skin of the external canal. The eruption consists of a crop of small vesicles which dries up after a few days. There is no specific treatment, apart from keeping the ear dry.
2. ***Herpes Zoster*** – Involving the geniculate ganglion may give rise to skin lesions, with or without involvement of the VII nerve resulting as Facial palsy (Ramsay-Hunt syndrome). The blisters usually dry up, leaving behind adherent crusts which persists for a week. Again, apart from keeping the ear dry no local treatment is necessary.
3. ***Bullous Myringitis (Otitis Externa Haemorrhagica)*** – Is characterised by the formation of blebs on the tympanic membrane and the deep meatal skin. The colour of the blebs is purplish due to hemorrhagic effusion filling the vesicles. The discharge won't relieve the pain (to differentiate from ASOM). Treatment consists of analgesics to relieve the pain, which is often quite severe. The ears is kept dry and antibiotics have no influence on the course of the disease. The blebs are not to be incised as this is of no value in relieving pain and it may introduce secondary infection.

C) FUNGAL INFECTION (OTOMYCOSIS)

Otomycosis is quite a common cause of otitis externa in the tropical climatic regions.

Etiology

1. ***Humidity*** – Occurs due to water entering the ear as a result of swimming or during a bath.
2. ***Infection*** – Caused by organisms Candida albicans, Aspergillus niger, Dermatophytes and Actinomycosis .
3. ***Antibiotic ear drops*** – Used over a long period lead to destruction of normally present bacteria resulting in growth of fungus.
4. ***Diabetics*** – Common
5. ***Immuno compromised individuals.***

Symptom

i. Itching in the canal is severe
ii. Pain is intense
iii. Deafness due to the blocked canal
iv. Ear discharge is blackish or brownish
v. Tinnitus may be present

Signs

i. Greyish white debris but black debris seen with Aspergillus
ii. Wet blotting paper appearance
iii. Features of Otitis externa

Treatment

1. The ears are cleaned for removing epithelial debris, as fungi thrive on them
2. Ear drops are given, which may be 2% salicylic acid in spirit, 1% clotrimazole or anti-fungi antibiotics like nystatin
3. Antibiotics are given only in the presence of secondary infection
4. Analgesics and anti-pruritic agents may be given

Treatment is continued for a week after the condition has apparently cleared up clinically.

II. REACTIVE OTITIS EXTERNA

A) ECZEMA

The eczematous reaction occurs as a result of sensitization of the skin.

Etiology

i. Infection
ii. Allergy
iii. Antibiotics ear drops e.g. neomycin.

Treatment

1. Local application of steroids
2. Antihistaminics
3. Ear is kept dry.

B) SEBORRHEIC DERMATITIS

This is a scaly condition of the scalp, referred to as dandruff.

Often it is associated with scaling in the external auditory canal, post-auricular sulcus and below the lobe of the pinna. The etiology is unknown.

Treatment

1. Regular removal of debris from the canal.
2. Washing hair with a shampoo for keeping the dandruff under control.
3. Steroid-antibiotic ear drops.

C) NEURODERMATITIS

In some cases of otitis externa there is an underlying psychosomatic disturbance which makes the condition difficult to clear up.

The initial symptom is irritation in the ear but on examination the skin is normal in appearance. However, constant scratching leads to lichenification or secondary infection of the skin causing a diffuse Otitis externa.

Treatment

i. Consists of clearing of secondary infections and attempting to alleviate irritation with topical hydrocortisone cream.
ii. In severe cases it may be necessary to bandage the ears to prevent scratching.
iii. Due attention must be paid to the psychological aspect.

WAX

Wax (cerumen) is a mixture of the ceruminous and sebaceous glands in the cartilagenous part of the external auditory canal.

The quantity of wax production varies greatly from individual to individual. In majority of the people wax dries up and separates as small flakes which are expelled from the canal, aided by the movements of the jaw.

Factors leading to wax impaction

1. Excessive production of wax or desquamation of epithelium
2. Change in its chemical composition
3. Narrow external canal or exotosis of canal
4. Dusty environment
5. Stiff hairs.

Symptoms

i. Pain is the commonest cause of earache
ii. Deafness due to mechanical obstruction
iii. Tinnitus
iv. Giddiness
v. Itching.

Sign

Wax is seen as brownish or a blackish mass filling the external canal.

Treatment

1. *Wax removal*
   a) Done with the help of a ring curette or wax hook. The instrument is passed distal to the wax and is then pulled out with usthe wax.
   b) Syringing is done when wax appears soft or is softened by wax dissolving agents.
2. *Softening agents*
   a) Oil was used in the past but is given up in favour better agents.
   b) Sodium bicarbonate ear drops are instilled four times a day for 3-7 days.
3. *Analgesics* – Are given in case of pain.
4. *General anaesthesia* – Is required for removal of wax which remains impacted even after using sodium bicarbonate ear drops.

SYRINGING

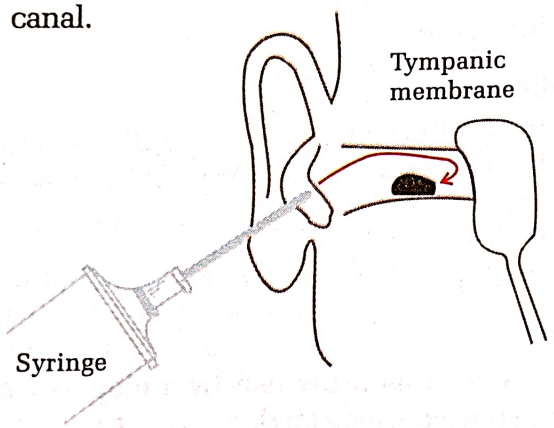
The aural syringe has a cylinder with a piston that can push water with force through a nozzle.

Indications

1. Removal of wax
2. Epithelial debris and dried crusts are removed
3. Foreign body removal

Contradictions

1. Hygroscopic foreign bodies, as they swell up and get further impacted.
2. Perforation of the tympanic membrane with otitis externa.
3. History of trauma or fracture of the external canal.



Ear syringing technique

Procedure :

1. The syringe is held in the right hand with the thumb into the central ring and the index and middle fingers into the other rings of the syringe.
2. Tap water is generally used after it is boiled and cooled down to body temperature. The use of hot or cold water would stimulate the labyrinth and result in giddiness (as in caloric test). Normal saline or distilled water can also be used for syringing.
3. The patient sits in the upright posture. A child is held firmly by the mother with legs of the child between the legs of the mother, one hand of the mother holds the child's hands while the other keeps the head fixed. A kidney tray is held by the assistant below the ear to receive the returning fluid from the syringe and the patient is covered with a towel to prevent clothes from getting wet.
4. The pinna is pulled upwards and backwards in adults while downwards and backwards in children.
5. The direction of the nozzle is directed towards the posterior wall or roof of the meatus.
6. The piston is gradually pushed and water flows at a steady rate. Forceful syringing may damage the tympanic membrane.
7. If wax totally occludes the canal a small opening along the wall is created by a wax hook, which now allows water to enter in and push out the wax.
8. The ear is made dry with a swab stick and then the tympanic membrane is examined.

Complications

1. *Trauma* to the external canal or tympanic membrane.
2. *Giddiness* due to use of hot or cold water.
3. *Infection* may be introduced by the entering fluids into the middle ear.
4. *Vasovagal* shock.

Malignant Otitis Externa

It is a fatal infection of the ear canal found in elderly, debilitated, uncontrolled diabetic patient's and sometimes in young, nondiabetic immunocompromised patient's.

It may affect the temporal bone, parotid gland, temporomandibular joint, skull base and cranial nerves. It is caused by the pseudomonas aeruginosa. Its enzymes destroy the local tissues. It presents as blood stained discharge. On otoscopy one can see a granulation tissue in the canal.

Most of time the infection is limited to external canal. It does not spread to middle ear. Tympanic membrane and mastoid is intact. In other words it is the Osteitis of temporal bone. The junction of cartilaginous and (8 mm) and bony (16 mm) part is involved.

Predisposing factors :

Elderly uncontrolled diabetics
Arteriosclerosis ,
Immunosuppression by Chemotherapy
High dose Steroid administration
Hypogammaglobulinaemia
Immunosuppressive disorders

Investigations :

Complete blood count
Blood sugar level
Renal function tests
Serum Electrolytes
Culture sensitivity of ear discharge
X-ray Mastoid (destruction of temporal bone)
Computerised Tomography
Magnetic Resonance Imaging (soft tissue destruction)

Diagnosis :

Bone scan

Differential diagnosis : Diffuse otitis externa

Treatment :

1) Control the Diabetes
2) Start systemic broad spectrum antibiotics (Over 6 to 12 weeks)

If penicillin sensitive, use Cephalexin.

Carbenicillin 24 mg IV daily
Gentamycin 3-5 mg/kg IM tid (7.5 mgm/kg in divided doses)
Local Gentamycin drops .

3) Surgical debridement of necrotic tissue.
4) In cases of complications one has to treat the underlying pathology (Like Parotidectomy if parotid is involved, Mastoidectomy if mastoids are involved)

It is a TOILET surgery, where only diseased cartilage is removed.

The best is prevention of this dreaded disease by control of diabetes.

Complications :

Facial palsy
Thrombosis of sinuses (Lateral, Superior, Inferior petrosal sinuses)
Osteomyelitis of base of skull
Cranial polyneuropathy (Involvement of IX, X, XI and XII nerves)
Cavernous sinus thrombosis
Mastoiditis
Meningitis

Prognosis : Surgically poor prognosis.

TUMOURS OF THE EXTERNAL CANAL

I. Benign

i. Papilloma
ii. Adenoma
iii. Exostosis
iv. Lipoma
v. Fibroma
vi. Chondroma, angioma etc.

II. Malignant

i. Squamous cell carcinoma
ii. Basal cell carcinoma
iii. Adenocarcinoma

Papilloma – Are caused by viruses, found in the external auditory canal.These are treated by curettage, local excision or Laser ablation.

Adenoma – Arise from sebaceous or ceruminous glands of external auditory canal. Sebaceous adenomas are painless, smooth tumours of the meatus and treated by local excision. Ceruminoma (Hidradenoma) are smooth polypoidal tumour in the canal. The tumour completely blocks the ear canal thus preventing any discharge which is treated by local excision. If not treated may develop into malignant adenocarcinoma.

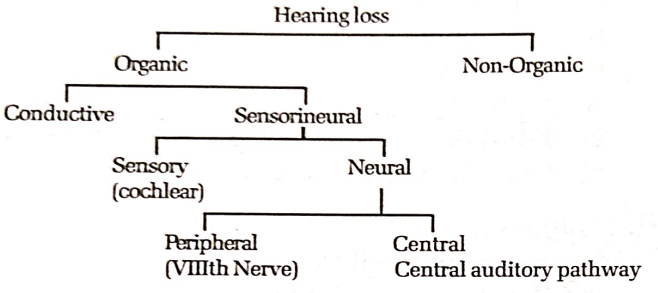
Exostosis – Are benign smooth bony growth in the bony external auditory canal. These are formed by dense ivory bone, being covered by normal skin. Most commonly occur in the swimmers, so refered as swimmers ear and treated by excision.

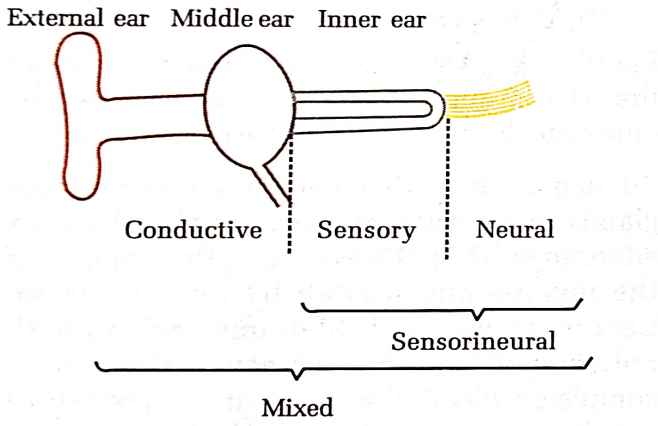
Squamous cell carcinoma – Occur in the pinna, external auditory canal or middle ear. Chronic otitis externa (discharging ear) is a predisposing condition. It involves neighbouring structures like temporal bone, cranial nerves and base skull. It presents as bloody discharge with foul smell, severe earache and headache associated facial paralysis and local lymph node involvement. Surgical excision of mass followed by Radiation is the treatment.

CHAPTER 7
Deafness

Deafness is defined as loss of hearing ability.

Depending upon the site of disease, the hearing loss or deafness may be classified as :





Types of Hearing Loss

Types

1. *CONDUCTIVE HEARING LOSS* – May occur due to disease affecting either the external ear or middle ear i.e. affecting the sound conducting mechanism of the ear. The common conditions are :
   i. ***Congenital*** – Atresia of the external canal, ossicular deformities ie. fixation of malleus head or stapes footplate, congenital cholesteatoma or ossicular discontinuity. Absence of oval window.
   ii. ***Traumatic*** – Foreign bodies or unskilled attempts for their removal, ossicular disconnection or perforation of the tympanic membrane after a blast.
   iii. ***Inflammatory*** – Otitis externa or following eustachian tube catarrah leading to otitis media.
4. ***Neoplastic*** – Exostosis (bony tumours) of the external canal or any malignant tumour involving the external or middle ear.
5. ***Miscellaneous*** – Wax or otosclerosis.

   The conductive hearing losses can be treated (reversible) resulting in a complete closure of the air-bone gap as seen in a puretone audiogram.

AVERAGE HEARING LOSS :

1. Complete obstruction of ear canal	30dB
2. Perforation of tympanic membrane	10-40dB
3. Ossicular interruption with intact drum	54dB
4. Ossicular interruption with perforation	38dB
5. Closure of oval window	60dB

Management

Most cases of conductive hearing loss can be managed by medical or surgical means. Treatment of these conditions is discussed in respective sections. Briefly, it consists of :

1. ***Removal of canal obstructions,*** e.g. impacted wax, foreign body, osteoma or exostosis, keratotic mass, benign or malignant tumours, meatal atresia.
2. ***Removal of fluid.*** Myringotomy with or without grommet insertion.
3. ***Removal of mass from middle ear.*** Tympanotomy and removal of small middle ear tumours or cholesteatoma behind intact drum.
4. ***Stapedectomy,*** as in osteosclerotic fixation of stapes footplate.
5. ***Tympanoplasty.*** Repair of perforation, ossicular chain or both.

6. ***Hearing aid.*** In cases where surgery is not possible,refused or has failed.

2. ***SENSORINEURAL HEARING LOSS*** – May occur due to involvement of the perceiving apparatus i.e. Cochlea or VIII nerve or anywhere upto the brainstem. The common causes being :
   i. ***Congenital*** – Birth trauma, prematurity, rubella during pregnancy, rhesus incompatibility etc.
   ii. ***Traumatic*** – Base skull fracture in a head injury (when it involves the labyrinth), noise induced hearing loss called 'acoustic trauma'.
   iii. ***Inflammatory*** – Various forms of bacterial and viral labyrinthitis, meningitis etc.
   iv. ***Neoplastic*** – Acoustic neuroma, tumours of the middle ear or nasopharynx invading the petrous bone or intracranial tumours involving the auditory pathway.
   v. ***Ototoxicity*** – Various drugs and chemicals can damage the inner ear (Table). Sensorineural hearing losses are permanent and when severe require rehabilitation by a hearing aid, lip reading, auditory training etc.

A. Aminoglycoside antibiotics -
   Streptomycin
   Gentamycin
   Tobramycin
   Neomycin
   Kanamycin
   Amikacin
B. Diuretics -
   Furesemide
   Ethacrinic acid
C. Antimalarials -
   Quinine
   Chloroquine
D. Cytotoxic drugs -
   Nitrogen mustard
   Cisplatin
E. Analgesics -
   Salicylates
   Indomethacin
   Phenylbutazone
   Ibuprofen
F. Chemicals -
   Alcohol
   Tobacco
   Marijuana
   CO poisoning
G. Miscellaneous -
   Erythromycin
   Ampicillin
   Propranolol
   Propyl thiouracil

SUDDEN SENSORINEURAL DEAFNESS

1. Infection Mumps & viral labyrinthitis
2. Trauma - Acoustic trauma, head injury, round window rupture.
3. Vascular - Spasm, thrombosis, embolism, hemorrhage.
4. Meniere's disease.
5. Central nervous system - Meningitis, trauma, encephalitis, vascular lesions.
6. Ototoxity - drugs mentioned above
7. Functional.

CHILDHOOD HEARING LOSS

Causes :

I. Prenatal

A) Genetic
   i. Waardenburg's syndrome
   ii. Klippel - Feil syndrome
   iii. Pendred's syndrome

B) Non-Genetic

1. Diseases during pregnancy
   i. Toxaemia
   ii. Diabetes
   iii. Nephritis
   iv. Measles and other viral infections

2. Drugs taken during pregnancy
   i. Streptomycin
   ii. Quinine
   iii. Salicylates

II. Perinantal
   i. Birth trauma - anoxia
   ii. Hemolytic disease - kernicterus
   iii. Prematurity

III. Postnatal

A. Genetic
   i. Otosclerosis
   ii. Familial perceptive deafness

B. Non-Genetic
   i. Infectious diseases e.g. measles, mumps etc.
   ii. Trauma
   iii. Otitis media
   iv. Ototoxic drugs

Deaf Mutism

The word itself is a misnomer as mutism is secondary to deafness which can be prevented. Deafness is not appropriate as more than 90% of these patients have some amount of residual hearing and thus require only amplification of sound for hearing.

Management

Conductive hearing losses that occur can be treated medically or surgically. Sensorineural hearing losses require amplification of sound (hearing aid), which if not beneficial then manual methods for communications have to be taught.

Presbycusis

Presbycusis is a condition in which hearing loss is associated with aging (degenerative changes), the hearing loss is sensorineural. Hearing loss increases from the age of 40 years to 75 years systematically.

Socioacusis is defined as hearing loss produced by exposure to nonoccupational noise in combination with life style factors such as diet and exercise.

Nosoacusis – Is the hearing loss attributable to diseases with ototoxic effects.

The hearing handicap is defined as the average hearing loss at 0.5 to 3 kHz must exceed 25 dB to be considered a hearing handicap .

Types of presbycusis -

1) Sensory presbycusis
2) Neural presbycusis
3) Strial presbycusis.
4) Cochlear conductive presbycusis.

The hearing loss is a slowly progressive, symmetrical sensori-neural hearing loss.The hearing loss is more in in the frequencies of 2000 Hz and speech discrimination is decreased.

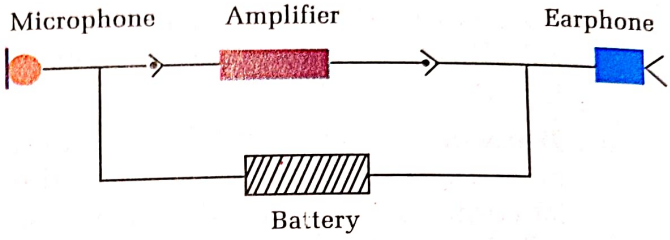
Patients can hear the sound but cannot interpret the words.

TREATMENT - Reassurance.

HEARING AID

It is a device which amplifies the sound and thus improves the hearing ability of the patient.

The performance of the aid depends upon the performance of each of its component i.e. microphone, amplifier and earphone.

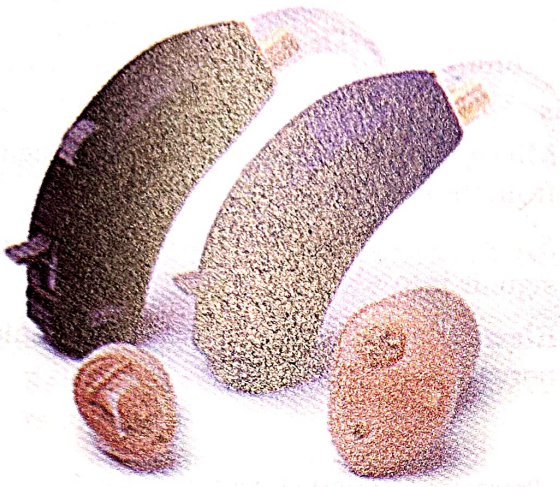


Hearing aid

Parts :

1. ***Microphone*** – Is made into a very small size and fits into the aid. Its function is to collect sound and convert it into electrical energy.
2. ***Amplifier*** – Magnifies these electrical impulses. The performance of a hearing aid amplifier is described in terms of amplification of the sound in decibels. A low frequency cut (high frequency emphasis) is useful as a means of reducing the effects of ambient noise and improving speech intelligibility.
3. ***Earphone*** – Acts as a speaker, whereby converting the magnified electrical energy to sound. Certain patients prefer bone conduction aids i.e. having a vibrator in place of a earphone.
4. ***Power supply*** – For the above mechanism is supplied by either 'penlight cells' or mercury cells.
5. ***Earmoulds*** – Allow the acoustic output from the hearing aid to be introduced into the ear, its functions are to
   i. Hold the earphone in place.
   ii. Act as a seal thus preventing an acoustic feedback (whistling sounds).

iii. Modify the acoustic characteristics (frequency and resonance of sound) of the aid by changing the ear mould dimensions.



Types of hearing aids

Types

1. ***Body worn type or Body aids*** – Is the most widely used aid, which is placed in the pocket of the vest and a wire conducts sound to the earphone. The advantages being its low cost and high amplification of sound obtained. These give high out-put for patients with profound hearing loss. But the rubbing of clothes against the microphone cause excessive noise.

2) ***Postaural type*** – Or behind the ear (BTE) - fits behind the pinna and is not visible as is covered by hair. The user can have flexible adjustment, due to their multiple control of electroacoustic properties.

3) ***Spectacle type or Eye glass aids*** – Aid gets attached to the spectacle frame.

4) ***In the ear type*** – **(ITE)** is inserted into the external auditory canal. These aids are very expensive and the amplification of sound obtained is limited. Cosmetically good as it is inside the ear and sound localization is good with it.

5) **In the canal aids (ITC)** – Cosmetically better than other aids. Its microphone improves the sound localization. It is good for patients with mild to moderate hearing loss. This fragile aid has limited adjustments due to its small size.

6) **Completely in the canal hearing aid (CIC)** – These are micro mini-aids which are placed deep in the canal (not easily visible). Patients can use telephone without hinderance. This solves the wind - noise problem. Can be placed securely in the ear and sound localization is good. It can be used in mild to moderate hearing loss. It cannot be used in abnormal (unfavourable) external auditory canals.

7) **CROS aids** – CROS stands for Contralateral Routing of Signals. These aids are used in individuals with minimal hearing or very poor hearing or unaidable hearing in the other ear. In this the microphone is placed on poor side of ear. The microphone receives the signals and after amplification routed to opposite ear by electrical cord or wireless FM radio signals. These aids help to hear sounds originated on the poor side.

8) **Bone anchored hearing aids (BAHA)** The bone anchored hearing aids are used in severe conductive hearing loss and congenital aural atresia patients. The child can be implanted as early as 2 and 3 years of age. This is a combination of bone conducting hearing aid and a Branemark titanium implant.

Advantages

1. Binaural and quality of voice is good.
2. Cosmetic and comfortable to wear.
3. Many times also helps in Sensorineural hearing loss.
4. Superior to conventional bone conducting hearing aids (specially in noisy environments).

Disadvantages

1. Expensive
2. Loss of fixtures (in 10% children)
3. Long term skin and septic complications

Rehabilitation of the deaf

This is done for patients having aquired hearing losses and not benefiting by the use of a hearing aid.

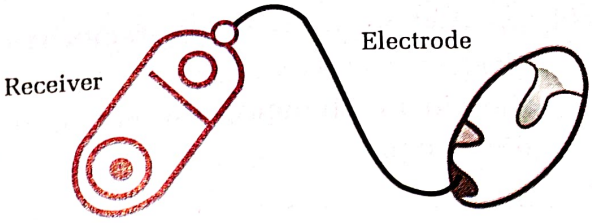
1. **Lip reading** – Can be described as a total process of looking at, perceiving and interpreting spoken words.
2. **Auditory training** – Includes noticing facial expression, intonating gestures as well as attending to acoustic clues. The auditory perception is based upon the ability to respond differently to different sound patterns.
3. **Finger spelling** – Is the simplest form of representing letters of the alphabet by the use of hands.
4. **Traditional signs** – Were used in the past between teachers and the children.

COCHLEAR IMPLANT

Cochlear Implant is a device which directly stimulates the auditory nerve with the conversion of sound energy into electrical energy thus they replace the non-functioning transducer system of hair cells of the cochlea. Unlike hearing aids which make sound louder, cochlear implants stimulate the auditory nerve electrically and thereby produce a sensation of sound in a different way.

Mechanism :

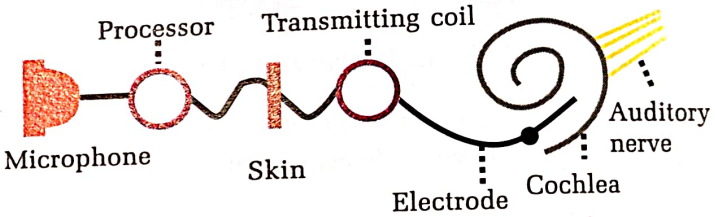
The electrode is inserted into the scala tympani via the round window. The sounds are picked up by an antenna in the transmitter which is worn like a hearing aid. The use of single channel cochlear stimulation did not lead to the transmission of intelligible speech and thus led to the development of multichannel electrode systems.



Cochlear implant

Parts

1. ***Microphone*** – Collects the acoustic signals from the environment.
2. ***Speech processor*** – Converts sound signals into electrical energy.
3. ***Electrode*** – That stimulates the cochlear nerve.



Cochlear implant

Types of Implant

1. ***House model*** – Is a single channel intra-cochlear implant, developed by William House. The electrode is inserted around 6 mm into the scala tympani. The speech processor uses an amplitude-modulated signal which is picked up by an induction coil placed under neath the skin.
2. ***Nucleas model*** – The advantage being, it is a multi-channel intra cochlear device. They consist of 22 electrodes that are inserted into the scala tympani. The speech processor transmits a code that is picked up by a micro-chip in the coil placed under the skin.

Indications

The various forms of aural pathology causing a total or very severe hearing loss.

1. Congenital deafness
2. Trauma - surgical, head injury etc.

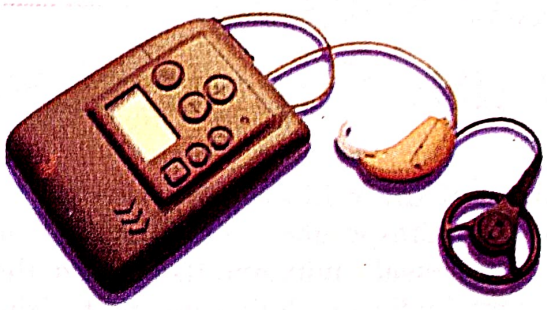
3. Labyrinthitis
4. Meniere's disease
5. Ototoxic drugs

These are the patients having a sensorineural hearing loss causing complete bilateral deafness. Better results are seen with those patients having an aquired deafness late in life, as they have developed and retained understanding of spoken means of communication.

Limitations of cochlear implant

1. Useful only in cochlear (sensory) hearing losses, when the retro - cochlear portion (auditory pathway) is normal.
2. Benefits post lingually deaf children.
3. Regular standarisation of the device is required.
4. A very expensive procedure.

Types of implants



Pocket worn

Post-Aural

CHAPTER 8

Acute Suppurative Otitis Media

The lining of the middle ear spaces is an extension of the respiratory mucous membrane that lines the nasal cavity and its sinuses, the nasopharynx and eustachian tube, part of the larynx, trachea and bronchi, down to the smallest terminal bronchiole. Acute Suppurative otitis media is nearly always a sequel to upper respiratory infection.

Etiology

Organisms reach the middle ear

1. Via the eustachian tube following nasopharyngitis, rhinitis, sinusitis, tonsillitis, pharyngitis etc.
2. Through a perforated tympanic membrane due to a previous disease, trauma or tympanotomy.
3. And very rarely by blood-borne infections.

Acute suppurative otitis media is seen commonly during childhood.

In infants where the eustachian tube is relatively short , wide and horizontal, milk and vomitus can easily enter the middle ear if the child is lying down while feeding or vomiting. In older children forcible nose blowing may have the same effect. (The eustachian tube is floppy and contains less elastin in children making children more prone to recurrent otitis media).

Organisms

1. Commonly found organisms are streptococcus hemolyticus, staphylococcus pyogenes and hemophilus influenza.
2. Viruses like measles prepare the ground for secondary invasion of the middle ear by these bacteria.

Pathogenesis :

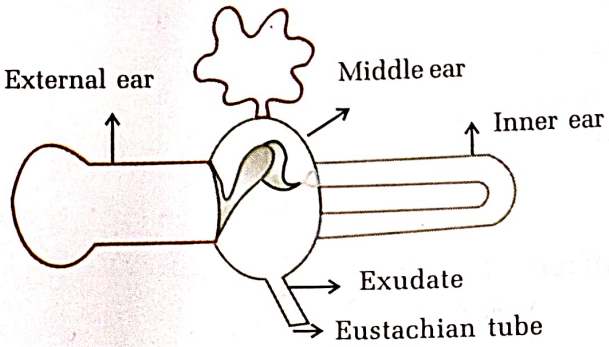
It usually occurs after an upper respiratory tract infection and commonly both ears are affected. The result is mucosal edema, paralysis of ciliary action and blockage of the eustachian tube. There is an increase in the number of goblet cells and absorption of gases produces a negative pressure in the middle ear. This retracts the tympanic membrane and promotes fluid exudation. If left untreated the increasing intratympanic pressure interferes with blood supply to the tympanic membrane and causes pressure necrosis resulting into a perforation. Ear discharge will continue till inflammation in the middle ear is settled, after which the tympanic membrane heals with a scar and hearing would return to normal.

Characteristics

1. It runs a characteristics clinical and pathological course with successive stages that are recognised by particular symptoms and otoscopic findings of each stage.
2. From the onset it tends to be a self-limiting disease.

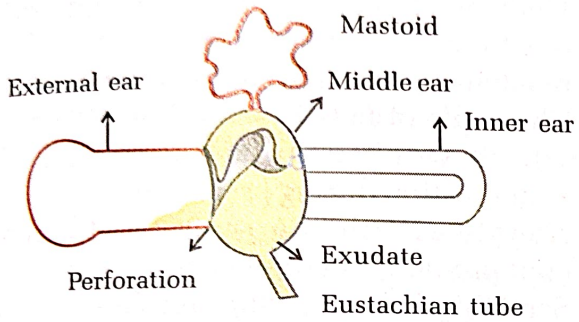
STAGES

1. ***Stage of Hyperemia*** – Is the first reaction by the middle ear space mucosa. The patient complains of earache and sense of fullness in the ear. Ear examination reveals congestion of the tympanic membrane due to dilated blood vessels. Therapy consists of nasal decongestants to re-establish the patency of the eustachian tube.



Stage of Exudation

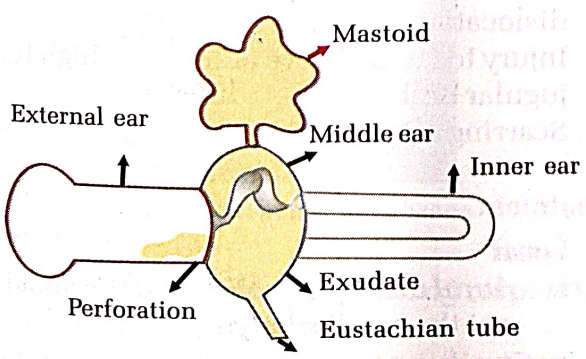
2. ***Stage of Exudation*** – Occurs due to outpouring from the dilated vessels into the middle ear spaces. Increased earache and fever are the symptoms. The ear drum is red and bulging. Therapy includes antibiotics and analgesics with nasal decongestants, failing which a myringotomy may be done.



Stage of perforation

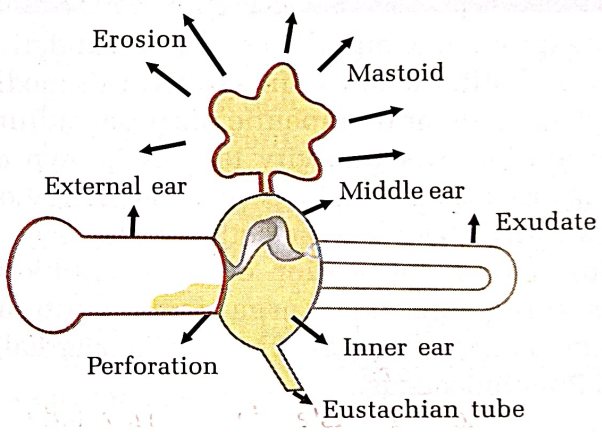
3. ***Stage of Suppuration*** – Results from release of exudate under pressure leading to a perforation in the tympanic membrane. Now pain is relieved but mild deafness is present. Culture and antibiotic sensitivity can be performed with the ear discharge and antibiotics are continued along with nasal decongestants. Ear drops even at this stage have no curative value as they do not reach the site of infection. Eustachian tube inflation is of no value until the acute inflammation begins to subside.

The perforation is most commonly seen in antero-inferior and postero-superior quadrants (in 85% and 15% of cases respectively).



Stage of Coalescent Mastoiditis

4. ***Stage of Coalescent Mastoiditis*** – Is the result of accumulation of pus under pressure in the mastoid air cells causing venous stasis, local acidosis and dissolution of calcium from the adjacent bony walls (halisteresis). As a consequence, separate air cells coalese into larger filled cavities full of purulent exudate and granulations. Symptoms being more severe pain, fever and increasing ear discharge. Radiography of the mastoid shows cloudiness of the air cells and a loss of distinctness of their cell partitions. Antibiotic therapy is continued, unless a fluctuant abscess is formed which would require drainage.



Stage of complications

5. ***Stage of Complications*** – Occurs due to extension of infection beyond the mucoperiosteal lining of the middle ear and pneumatic air cells to adjacent structures leading to complications. This may manifest as a postaural abscess or an extension to the facial nerve or the labyrinth, an extradural abscess, sigmoid sinus phlebitis, brain abscess or meningitis may occur. Complication by coalescent bone erosion calls for antibiotic therapy combined with surgical evacuation of the abscess cavity.

6. ***Stage of Resolution*** – Comprises of healing of the tympanic membrane with normal hearing. This occurs following an effective antibiotic medication or host resistance

overtaking the virulence of infecting organisms.

The effusion will persists for a period of 3 months in 70% of cases.

Investigatons :

1. ***Ear discharge*** – For culture and antibiotic sensitivity.
2. ***X-ray mastoid*** (Schuller's view) – To detect the destruction of air cell partitions.
3. ***Hearing test*** – To detect conductive hearing losses. (Tuning fork and Pure-tone audiometry).

TYMPANOCENTESIS

The aspiration of middle ear fluid in exudative phase of otitis media or in serous otitis media for diagnostic or therapeutic purpose (culture and antibiotic sensitivity test) is known as Tympanocentesis. This helps in the identification of causative agent and selection of the drug. It helps in the identification of newer antibiotic -resistant bacterial organisms such as Haemophilus influenzae or Moraxella catarrhalis and Pneumococcus.

Indications

1) A patient of Otitis media with severe earache or toxic.
2) Those who are not responding to antibiotics or failure of antibiotic treatment.
3) Otitis media patients who are prone for complications.
4) In immunonodeficiency, sick neonates or new-borns with otitis media.

Pre-requisites

1. History of ear problem
2. Otoscopic ear examination
3. Assesment of tympanic membrane mobility with pneumatic otoscope
4. Impendence audiometry

Procedure

Patient is sedated or general anaesthesia given in infants.

- Turn the head to opposite side i.e. affected ear will be above.
- Clean the canal completely. Gently swab the ear canal with alcohol soaked applicator as a "wet-mop".
- 1ml Tuberculin syringe with 22 or 23 gauge, 2½ to 3 inch spinal needle (Adults 18 no.) used.
- Under microscope aspiration of fluid carried out from antero-inferior or postero-inferior quadrants of maximal bulge for 1to2 seconds.
- The angle of needle should be 30° or 45°, which won't hamper the vision (for visualization during procedure).
- Needle should be removed to avoid contamination in the canal.
- Send this fluid for culture and Gram's stain.
- Keep sterile cotton in the ear. Antibiotic soaked gelfoam kept.
- (Normally perforation heals within 3 to 5 days).
  (Swimming prohibited)

One should not use Nitrous anaesthesia while doing Tympanocentesis, as it displaces the middle ear fluid. If thick glue present, it is not possible to do aspiration, then one should go for myringotomy.

Complications

- Middle ear infection (as direct communication established in between external and middle ear)
- Perforation of tympanic membrane
- Persistent perforation
- Damage to middle ear structures like injury to ossicles (Incudo-stapedial joint dislocation)
- Injury to fascial nerve or Injury to high lying jugular bulb
- Scarring of tympanic membrane

Treatment

A) Local

i. *Aural toilet* – Is maintained by sucking out the ear discharge.

ii. *Boro-spirit* – Ear drops are used only after a perforation occurs. Ear drops are

not recommended before a perforation as they are unable to reach the middle ear and they may obscure the otoscopic picture.

B) Systemic

i. *Antibiotic* – Therapy is the most important form of treatment and must be used in all stages of disease.

ii. *Nasal decongestants* – Drops are used to improve drainage of the exudate via the eustachian tube. (Ephedrine, Oxymetazoline or Xylometazoline nose drops).

iii. *Anti-histaminics* – Are given to decongest the middle ear mucosa, eustachian tube and paranasal sinus mucosa.

iv. *Analgesics* – To relieve pain.

C) Surgical

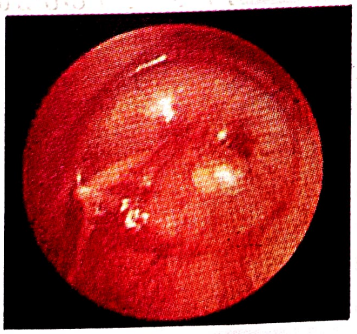
Myringotomy may be done only in (10%) cases of severe earache with a bulging drum or when impending intracranial complications present with an inadequate drainage.

MYRINGOTOMY

Definition

It is an incision made on the tympanic membrane to drain out the middle ear fluid.

The aim of this procedure is to confirm the diagnosis, to aspirate fluid and insertion of grommet, if needed.



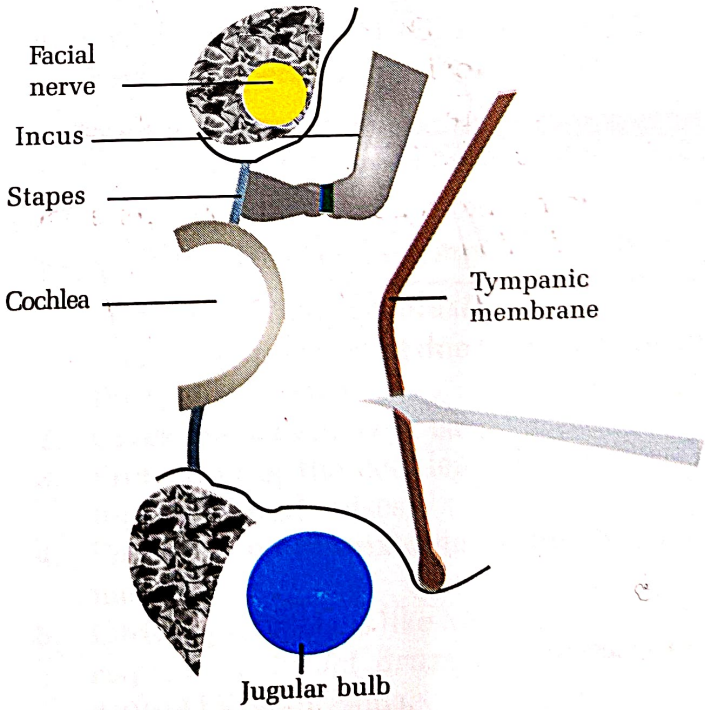
Bulging Tympanic Membrane

Indications

1. Acute otitis media , not having responded to medical line of treatment.
2. Impending intracranial complications in acute otitis media with inadequate drainage.
3. Secretory otitis media.
4. Haemotympanum.
5. Suspected malignant disease of nasopharynx (Aspirated fluid for cytology)
6 Chronic retraction pockets of the tympanic membrane or tympanic membrane atelactasis
7. Autophony secondary to Eustachian tube dysfunction
8. Barotitis media

Technique

1. Performed under local anaesthesia, unless in a child where General anaesthesia is used as slightest movement would lead to damage to other structures.
2. The external canal is cleaned with the preparing solutions.
3. An incision is made with a sharp instrument under microscopic vision in the postero-inferior quadrant, midway between the annulus and umbo. The site is chosen as here the blood supply is sparse and chance of damage to middle ear structures is the least.
4. The middle ear fluid is sucked out.
5. Incision site covered with gelfoam. No post-op management is necessary.



Myringotomy incision by Sickle Knife

Complications

1. Anaesthesia
2. Facial nerve damage
3. Incudo-stapedial joint dislocation
4. Chorda Tympani damage
5. Injury to a high jugular bulb
6. Round window damage
7. Perforation in the tympanic membrane
8. Introduction of infection into the middle ear.

SPECIAL FORMS OF ACUTE OTITIS MEDIA

1. **Infants :** There may be no symptoms related to the ear, but there may be fever, meningism and upper respiratory tract infection. It may be associated with gastro-enteritis. The drum is congested and edematous.
2. **Children :** The clinical features are same as in adults, but there may be high fever.
3. **Exanthemata** lower the resistance and there may be massive destruction of the ear drum and ossicles, due to acute necrotic otitis media.
4. **Masked Acute Otitis Media :** With inadequate antibiotics, the infection may be masked to a great extent and complications may develop silently.
5. **Mastoiditis (Diabetic Otitis Media) :** Sudden massive destruction may occur with few symptoms.
6. **Haemorrhagic Otitis Media :** With influenza epidemic, one may come across haemorrhagic exudate in the middle ear and haemorrhagic vesicles on the drum. Streptococcus haemolyticus is the usual organism and the pain is very severe.
7. **Infection by Pneumococcus Type 3 :** produces features similar to masked acute otitis media.

Myringoplasty - repair a hole in the ear-drum

Tympanoplasty - repairing of ear-drum + reconstruction of middle ear bones

CHAPTER 9

Chronic Suppurative Otitis Media

Chronic Otitis Media is chronic infection of the middle ear cleft, can be of types :

(>3 months duration)

1. Perforation of Tubo-tympanic or Attico-antral type.
2. Chronic non-suppurative otitis media eg. Serous otitis media.
3. Chronic specific otitis media eg. following tuberculosis.

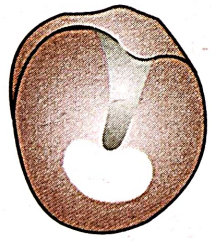
Types :

I. TUBOTYMPANIC DISEASE (SAFE TYPE)

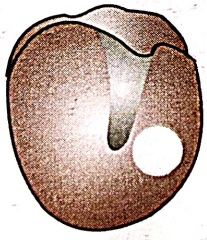
Etiology :

i. Residue of an acute otitis media infection during childhood.
ii. Repeated infections from the eustachian tube.
iii. Large sized traumatic perforation.

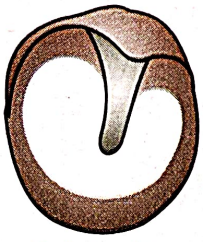
Perforation – Usually in the form of a central perforation situated exclusively in the pars tensa. The ossicular chain often remains intact and middle ear mucosa is pink and edematous.



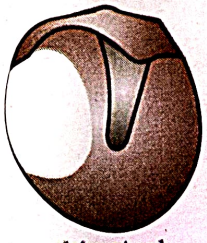
Central perforation (moderate)



Central perforation (small)



Sub total perforation



Marginal perforation

Clinical features :

1. ***Discharge*** – is mucoid, copious and non foul smelling. It becomes profuse during an upper respiratory infection.
2. ***Deafness*** – is conductive in type. Degree varies with the position and size of perforation.

Treatment:

1. *Aural toilet* – Is performed meticulously under direct vision, preferably suction and irrigation done under the operating microscope.
2. *Boro-spirit* – Ear drops are advised only during active discharge.
3. *Focus of infection* – Are treated medically or surgically e.g. tonsillitis, sinustitis etc.
4. *Removal of polypi or granulation tissue* – if present in the middle ear.
5. *Chemical cautery* – Using 50% Trichloro acetic acid for smaller perforations (< 65% area if tympanic membrane).
6. *Myringoplasty* – May be required for a persisting perforation.

MYRINGOPLASTY

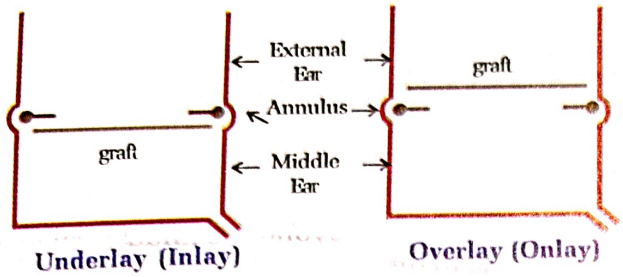
Definition

Repair in the defect of the tympanic membrane.

Advantages of Myringoplasty

1. Hearing improvement due to an increase in the tympanic area.
2. Gives the patient a dry ear.
3. Protection of the cochlea from the direct impact of loud noises.
4. Prevents an otitis externa leading to otitis media.
5. Certain professions like Mining and Flying require an intact drum due to damage caused by barotrauma.

Techniques



Underlay (Inlay) Overlay (Onlay)

The above methods differ in the technique of graft placement. Graft is placed lateral to the annulus in Overlay and medial to the annulus in underlay technique. However, the Underlay technique is preferred by most surgeons today. (Technique similar as in Tympanoplasty).

Advantages of Underlay technique

1. Quicker procedure.
2. Allows inspection of the middle ear.
3. No epithelial cells are left under the graft, so no danger of cholesteatoma formation.
4. After healing, the graft does not lateralise due to fibrosis.
5. The contour or angulation of the anterior tympanomeatal angle is maintained.

II. ATTICO - ANTRAL TYPE (Unsafe type)

It is associated with a cholesteatoma and therefore regarded as unsafe. The term cholesteatoma is not an accurate description as it is not a tumour and always does not contain cholesterin. The simplest definition of a cholesteatoma is 'skin in the wrong place' (Gray). This affects the postero-superior part of middle ear cleft.

Cholesteatoma is a three dimensional epidermal and connective tissue structure, usually in the form of a sac and frequently conforms to the architecture of various spaces of the middle ear and mastoid. This structure has the capacity for progressive and independent growth at the expense of the underlying bone.

(The word "Cholesteatoma" was coined by Johannes Mueller)

Classification :

1. ***Congential*** – originates as embryonic rest cells, which eats away the bone until it breaks through the outer attic wall.

Derlaki and Clemis defined congenital cholesteatoma or Primary choleasteatoma as an embryonic rest of epithelial tissue in an ear without tympanic membrane perforation, in a patient without history of ear infection.

2. ***Acquired :***

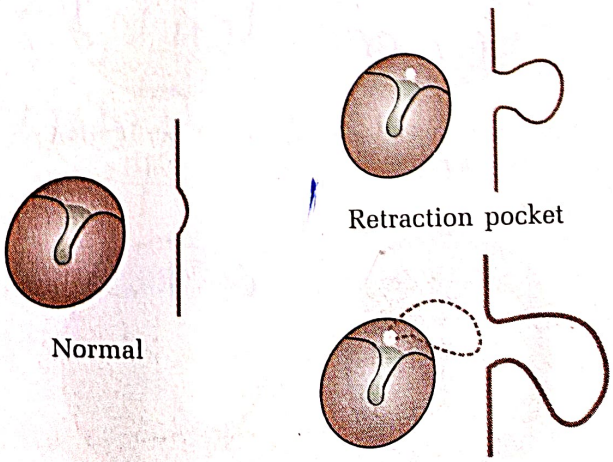
i. ***Primary*** – Type occurs following an eustachian tube malfunction resulting into retraction pockets into the attic (pars flaccida).

Negative middle pressure causing retraction pocket

ii. ***Secondary :***

a) ***Immigration*** – Of tympanic epithelium on deep meatal skin in the posterior superior quadrant of the tympanic membrane occurs. This is possible following destruction of the annulus after a previous middle ear discharge.

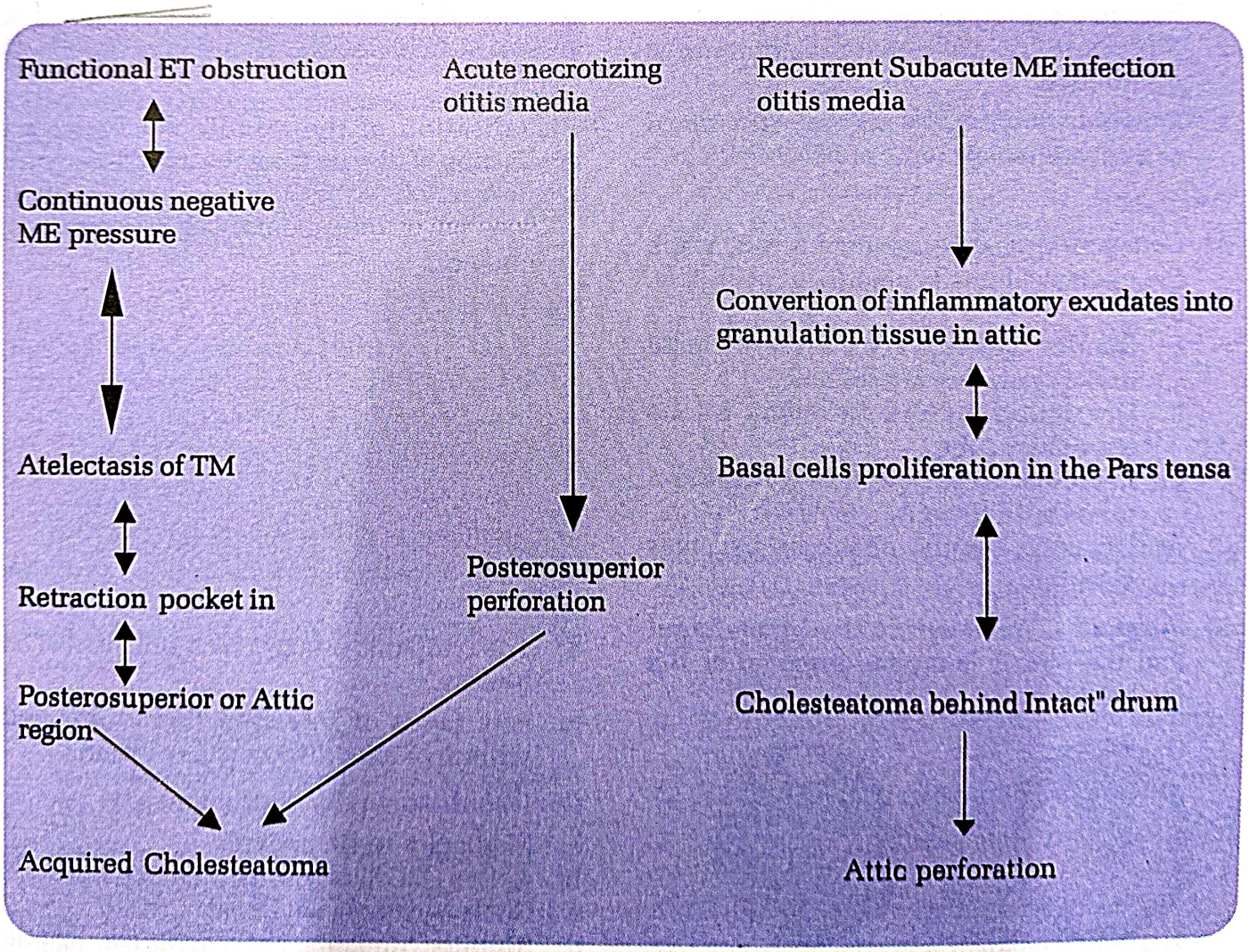
b) ***Metaplasia*** – Is provoked by chronic infection in the ciliated mucous membrane of the middle ear. Here squamous metaplasia occurs as well as there can be a marginal migration of squamous epithelium from the meatus.



Pathogenesis of cholesteatoma

1. The focal area is the pars flaccida, posterior superior part of pars tensa and skin of the deep external meatus.
2. Following eustachian tube obstruction, oxygen is absorbed by the middle ear mucosa producing intratympanic vaccum.
3. Transudation or exudation of fluid occurs creating surface tension on the undersurface of the tympanic membrane.
4. Under this stress, the pars flaccida gives way and forms a retraction pocket.
5. Keratin collects in the retraction pocket which becomes moist and thus an ideal medium for growth of proteolytic bacteria e.g. Proteus.
6. Next , ulceration of the epidermis lining the pocket occurs proceding to osteitis with bone resorption.
7. Expansion of the cholesteatoma occurs into the aditus, antrum, then into other mastoid air cells. In the process the ossicular chain is destroyed and the cholesteatoma has the potential to go beyond the middle-ear cleft leading to the various complications.

I) Secondary acquired cholesteatoma
II) Primary acquired cholesteatoma



Pathogenesis of Cholesteatoma

Clinical Features

1. ***Ear discharge*** – Is scanty, foul smelling and some times blood stained.
2. ***Deafness*** – May go unnoticed if a portion of the cholesteatoma may bridge the gap in the ossicular chain. With sensorineural hearing loss the patient may complain of deafness.
3. ***Complications*** – May cause symptoms of headache, vertigo, facial palsy or earache.

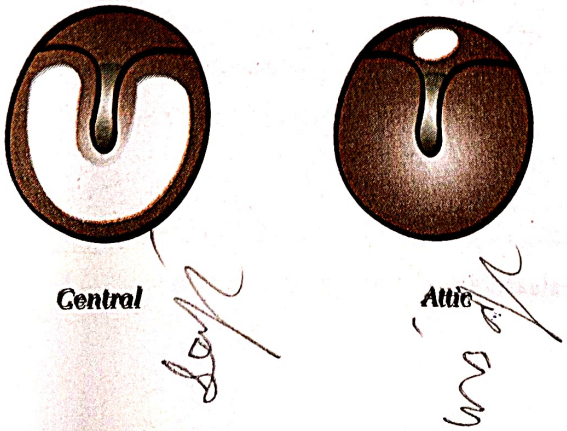
Diagnosis

1. ***Otoscopy*** – Perforation situated either in pars flaccida or marginally in the posterior superior quadrant of the pars tensa. Cholesteatoma is visible with the perforation and the greyish white or pearly sheets of kertain are easily recognised.
2. ***Radiography*** – Of the mastoid, shows bone destruction.
3. ***Puretone audiometry*** may reveal a mixed or a sensorineural type of hearing loss.

Treatment

A) ***Conservative*** – Management is tried when the cholesteatoma is small and accesible, by:
   i. Suction clearance of cholesteatoma under operating microscope.
   ii. Dry mopping and inflation of the eustachian tube.
   iii. Removal of polypi and granulation tissue with forceps.
   iv. Life long, regular check up becomes essential.

B) ***Surgical*** – Treatment is always preferred and the aim is complete removal of the cholesteatoma.



	SAFE EAR	UNSAFE EAR
i. Type of Disease	Tubo-tympanic	Attico-antral
ii. Perforation site	Central	Attic
iii. Granulations	Absent	Common
iv. Discharge	Copious, mucoid, and non-foul smelling	Scanty, purulent and foul smelling
v. Bleeding	Never	Sometimes
vi. Respiratory tract infection	Increase in the amount of discharge	Not affected
vii. Audiogram	Conductive hearing loss	Mixed hearing loss
viii. X-ray mastoid	Sclerotic	Sclerotic with destruction
ix. Complications (Fatal)	Nil	Common

TYMPANOPLASTY

Definition

Visualisation of the middle ear cleft and eradication of disease from the middle ear with reconstruction of the ossicular chain, with or without repair of the defect in the tympanic membrane.

Pre-requisites for a tympanoplasty

1. Hearing must be better by bone conduction, meaning the patient must have a conductive hearing loss.
2. Patency of the eustachian tube is established.
3. Adequate cochlear function is present.
4. State of ossicular chain and labyrinthine windows must be assessed.
5. The tympanic mucous membrane must be healthy.
6. Any focus of infection like a deviated septum or tonsillitis must be treated.

Contraindications for tympanoplasty

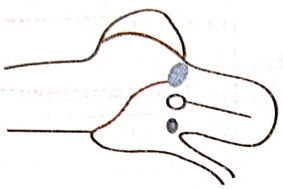
a. Medically unfit patients
b. Active CSOM or discharging Ear
c. Choleasteatoma i.e. Unsafe CSOM
d. Rhinitis, Sinusitis, Pharyngitis etc. (septic foci)

Types of Tympanoplasty

Type I (Myringoplasty)

Type II

Type III (Myringostapediopexy)



Type IV

Classified according to the pattern of sound transmission. It becomes very convenient to refer to these mechanisms when assesing an existing defect or when planning a reconstruction during surgery.

Type 1 - A normal middle ear with intact mobile ossicular chain.
Type 2 - A deformed but a functioning ossicular chain.
Type 3 - Transmits the sound directly through a mobile stapes.
Type 4 - Leaves the oval window with the mobile foot plate exposed.
Type 5 - Sound directly enters through a fistula into the lateral semicircular canal.
Type 6 - Sound Inversion. Here the sound enters the round window, keeping the oval window covered (direction reversed) (Sono-inversion).

Ossicular grafts :

I. ***Autografts***
   i. Ossicles e.g.Malleus head,Incus
   ii. Cartilage e.g. Tragal, Conchal, Septal
   iii. Bone e.g. spine of Henle

II. ***Homografts***
   i. Ossicles from Human cadavers
   ii. Cartilage
   iii. Bone e.g, costal cartilage, styloid process etc.
   iv. Human Dentine

III. ***Implants (Inert materials)***
   i. Stainless steel
   ii. Teflon
   iii. Sialastic
   iv. Ceramics
   v. Polythene
   vi. Gold
   vii. Calcium hydroxyapatite
   viii. Plastipore

Implants are of two types:

1. ***PORP (Partial Ossicular Replacement Prosthesis)*** used for ossicular reconstruction from ear drum to head of stapes.
2. ***TORP (Total ossicular Replacement Prosthesis)*** used for reconstruction from ear drum to foot plate of stapes.

Tympanic Graft materials :

I. ***Autografts***
   i. Temporalis Fascia
   ii. Tragal perichondrium
   iii. Extra-meatal skin.
   iv. Vein graft
   v. Conchal perichondrium
   vi. Fascia lata

II. ***Homografts (Human)***
   i. Cadaveric fascia
   ii. Tympanic membrane
   iii. Vein graft
   iv. Lyophilised dura
   v. Amnion

Temporalis fascia is the commonest graft used to cover the defect in the tympanic membrane.

Advantages :

1. Obtained from the same incision.
2. Autograft, therefore rejection chances minimal.
3. Obtained of any size and of no cost.
4. Thickness is the same as normal tympanic membrane.
5. Provides good resistance to infection.
6. Low metabolic rate, therefore remains in the live state throughout surgery.

Technique

1. Done under General Anaesthesia in children and unco-operative adults.
   Local anesthesia is however preferred.

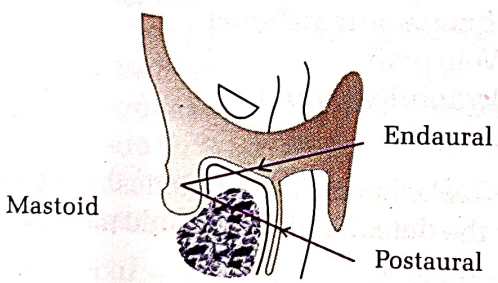
Advantages of Local anaesthesia :

i. Bleeding is minimal.
ii. Hearing results can be tested on table.
iii. Symptoms of facial paralysis or labyrinth stimulation are detected immediately.
iv. No side-effects of General anaesthesia.
   About 5cc of 2% Lignocine with 1 in 1 lac adrenaline is injected in the postaural groove and around the canal wall.

2. The part is painted, prepared and draped , taking care none of the preparing agents enters into the middle ear, as they can cause permanent sensorineural hearing loss.
3. A postaural incision is taken. This is 0.5 cm behind the postaural groove from the upper end of the pinna to the mastoid tip.

Advantages of postaural incision

i. Scar is not seen
ii. Temporalis fascia obtained from the incision site
iii. The same incision also allows a mastoidectomy
iv. No chance of perichondritis.



Surgical approach to the middle ear

4. The incision passes through skin and subcutaneous tissue. A mastoid retractor is applied at the upper end of the incision from where temporalis fascia is obtained after separating it from the temporalis muscle.
5. Now the incision is deepened so as to expose the mastoid bone. The periosteum is elevated anteriorly to expose spine of Henle and posterior canal wall on which a horizontal cut is made.
6. The edges of the perforation are freshened and undersurface of remnants of the drum are scrapped to make a raw surface. (Underlay)
7. Two vertical incisions, at 12 o' clock and 6 o'clock are made and the posterior canal skin is elevated till the annulus.
8. The annulus is dislocated anteriorly, middle ear inspected, disease removed and ossiculoplasty done according to the pathology present.
9. Defect in the tympanic membrane is covered by the temporalis fascia which is placed underneath the remants of the drum and the posterior canal skin.
10. Gelfoam laden with antibiotics is placed over the new drum and on the canal skin.
11. The periosteum and subcutaneous tissue is closed with catgut and skin with black silk.
12. Pressure dressing is given in the form of a mastoid dressing.

Complications

I. Immediate

i. Anaesthesia
ii. Facial paralysis
iii. Sensorineural hearing loss
iv. Labyrinthitis

II. Delayed

i. Dead ear
ii. Conductive hearing loss
iii. Graft rejection
iv. Perichondritis.

Tubercular Mastoiditis

The occurrence of Tuberculous Otitis media was a rarity but a resurgence is seen, as being associated with AIDS and always due to primary tuberculosis.

Presents as painless ear discharge, decreased hearing and sometimes Facial paralysis. On otoscopy multiple perforations are the classical feature of tubercular infections.

Chronic ear discharge not responding to medical management requires to rule out tuberculous involvement.

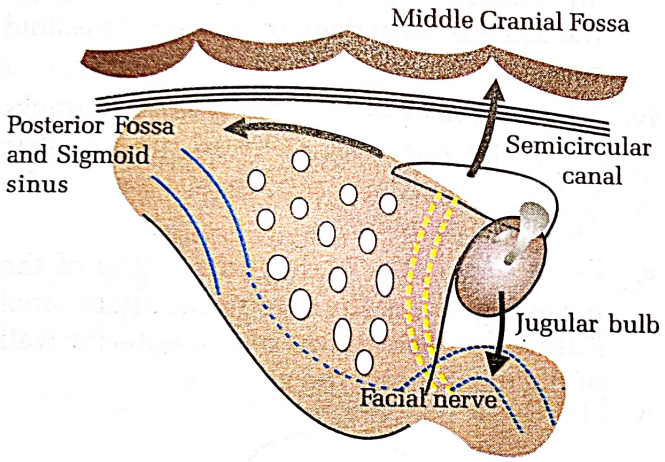
Management – Anti Tubercular treatment with ear toilet.

CHAPTER 10

Complications of Otitis Media

The safe type of central perforation does not by itself invade the bone and cause complications. The bone invading type of chronic otitis media seen with a cholesteatoma is responsible for the various intracranial complications.

Certain strains of hemolytic streptococci cause more complications than other strains of the same organism. Type III pneumococcus has a particular predilection for intracranial extension.



Routes of spread

Factors leading to complications

1. Poor patient resistance
2. Highly virulent organism
4. Inadequate therapy
5. Resistance to antibiotics
6. Immunocompromised patient.

Pathways of spread :

1. ***Osteothrombophlebitis*** – Occurs when infection passes from the lining of the tympanic cavity and mastoid air cells through intact bone by means of a progressive thrombophlebitis of small venules.
2. ***Bone erosion*** – is usually due to a cholesteatoma and rarely as a result of a chronic otitis media.
3. ***Preformed pathway*** – may be
   i. Abnormal anatomical opening such as the oval or round window leading from the middle ear to the labyrinth.
   ii. The internal auditory meatus, perilymphatic duct or endolymphatic duct and sac leading from the labyrinth to the meninges.
   iii. A developmental dehiscence, such as a patent petro-squamous suture in infants or a dehiscence of the floor of the hypotympanum over the jugular bulb.
   iv. Result of skull fracture or a previous surgery e.g. stapedectomy.
   v. A perilymph fistula may be congenital or acquired and establishes a wide opening for infection to extend to the labyrinth and hence to meninges, following an attack of otitis media.

   Extension by a preformed pathway is not always easily diagnosed pre-operatively.

The Complications are :

I. Non-Meningeal

1. Mastoid infections
   A. Mastoiditis
   B. Mastoid abscess
2. Petrositis
3. Facial paralysis
4. Labyrinthitis
5. Brain abscess

II. Meningeal

1. Extradural abscess
2. Subdural abscess
3. Lateral sinus thrombosis
4. Otitic hydrocephalus
5. Meningitis

I. NON-MENINGEAL COMPLICTIONS

1. MASTOID INFECTIONS :

A) MASTOIDITIS

a) ***Acute Mastoiditis*** – Results from an acute otitis media when
   i. there is a lowered resistance of the patient
   ii. Virulence of the organism is great
   iii. Drainage via eustachian tube or through the perforation is poor.
   iv. Improper inadequate antibiotics given
   v. Mastoid highly cellular (pneumatic)

b) ***Chronic Mastoiditis*** – When the cholesteatoma erodes mastoid bone and then the mastoid is filled with granulations and cholesteatoma. This is usually seen in sclerotic mastoids.

Clinical features

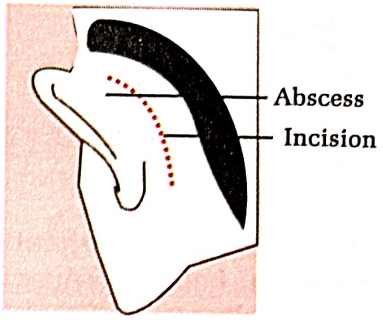
i. ***Pain*** – Occurs again, was present with acute otitis media.
ii. ***Ear discharge*** – Would increase in quantity.
iii. ***Tenderness*** – Is elicited over the mastoid antrum area.
iv. ***Sagging*** – Of the posterior superior bony canal wall.

B) MASTOID ABSCESS

Both forms of acute and chronic mastoiditis may result in necrosis of the bone and pus tracks out leading to different types of abscesses.

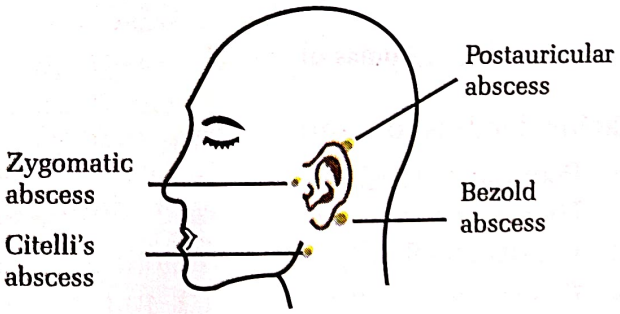
Mastoid cells when infected will form an abscess and need surgical intervention.Depending on their locations they are described as follows :

i. ***Subperiosteal abscess*** – Is the commonest abscess seen and is also known as post-auricular abscess. The pinna is displaced outwards, forwards and downwards.



Incision for post - Auricular abscess

ii. ***Zygomatic abscess*** – Forms when the pus tracks along the zygomatic process of the mastoid and causes a swelling in front of the ear, which may be confused with a parotid swelling.
iii. ***Von Bezold's abscess*** – Occurs following an erosion of the tip of mastoid process and the abscess form deep to the sternomastoid muscle.
iv. ***Citelli's abscess*** – Is seen when pus tracks along the posterior belly of the digastric muscle resulting into an abscess in the digastric triangle.
v. ***Luc's abscess*** – Is due to sagging of the posterior wall of the external auditory canal following destruction of the anterior wall of the mastoid by the abscess.



X-ray mastoid – shows clouding of the air cells with destruction of the intercellular partitions.

Treatment

a) *Acute Mastoiditis* –
   i. *Antibiotics*
   ii. *Myringotomy* for draining the exudate
b) *Chronic Mastoiditis* – Requires a mastoidectomy, the type of which would depend upon the extent of disease present.

2. PETROSITIS

It results from extension of infection into the body and apex of petrous bone.

Clinical features

i. Earache and profuse ear discharge.
ii. Unilateral headache with retro-orbital pain caused by trigemminal neuralgia.
iii. Sixth nerve paralysis.

Gradenigo's syndrome is a triad of ear discharge, pain around the eye and sixth nerve (VI) paralysis.

Treatment

A mastoidectomy is required along with clearance of disease from the petrous part. Antibiotics are a must.

3. LABYRINTHITIS

Can occur in various forms :

i. ***Perilabyrinthitis*** – Following formation of a fistula which threatens spread of infection into the inner ear.
ii. ***Serous labyrinthitis*** – Diffuse labyrinthine infection without formation of pus.
iii. ***Suppurative labyrinthitis*** – Which is also diffuse labyrinthine infection but is associated with pus formation.

Clinical features

Patient complains of nausea, vomiting, deafness, tinnitus and giddiness. The patient has sensorineural hearing loss and fistula test is positive.

Treatment

i. Strict bed rest Head movements are to be avoided.
ii. Labyrinthine / Vestibular sedatives given to control vomiting and giddiness. Eg, Prochlorperazine or Cinnarzine.
iii. Antibiotics form the primary line of treatment.
iv. If labyrinthitis is due to acute otitis media, one should consider myringotomy. Antibiotics may be changed on the culture report of aspirated pus.
v. Symptoms persisting, a mastoidectomy would be required for peri and serous labyrinthitis, while a labyrinthectomy for purulent labyrinthitis.

Once patient recovers from symptoms, Cawthorne - Cooksey exercises are advised to improve central compensation of labyrinth.

4. BRAIN ABSCESS

Occurs in the temporal lobe , twice as often in the cerebellum. As a rule otitic brain abscess are single, in rare cases may be multiple.

Clinical features

1. Fever, headache, nausea, vomiting or convulsions may be seen.
2. Aphasia occurs with a left temporal abscess in a right handed patient.
3. Paresis of the face and mouth on the opposite side, later involving the arm and hand due to the pressure on the motor cortex.
4. Visual disturbances may be detected with temporal lobe abscess.

Stages :

1. Initial cerebral thrombophlebitis , cerebral edema and encephalitis precede focal necrosis and liquefaction.
2. A capsule may be recognisable in 2 weeks but becomes thick by 6 weeks. Later the fibrous tissue of the capsule undergoes hyaline degeneration and calcification.
3. An abscess may rupture into the ventricle or subarachnoid space or an expanding abscess may lead to a rise in intracranical pressure causing tentorial herniation.

Investigations :

1. Fundoscopy – Revealing papilloedema.
2. Lumbar puncture – For CSF examination.
3. CT Scan – To detect the exact size and site of abscess.
4. CT Scan with Contrast (Conray) – Helps to know the size and location of abscess.
5. MRI Scan - Easily differentiate normal brain, edema of brain, pus and capsule of abscess.

Differential Diagnosis :

Meningitis
Subdural abscess
Sinus thrombophlebitis
Otitic hydrocephalus
Brain tumours

Treatment :

1. ***Drainage*** – Of the abscess by burr hole.
2. ***Antibiotics*** – In high doses.
   The intracranial pressure can be lowered by Steroids (Dexamethasone 4mg iv 6th hourly) and intravenous 0.5 gm/kg 20% Mannitol.
3. ***Mastoidectomy*** – Is required once the abscess has cleared.

II. MENINGEAL COMPLICATIONS

1. EXTRADURAL ABSCESS

It occurs due to spread of infection to the extradural space and such an abscess around the sigmoid sinus is called a perisinus abscess. The collection of pus is usually well encapsulated but may spread by stripping dura from the bone.

Clinical features :

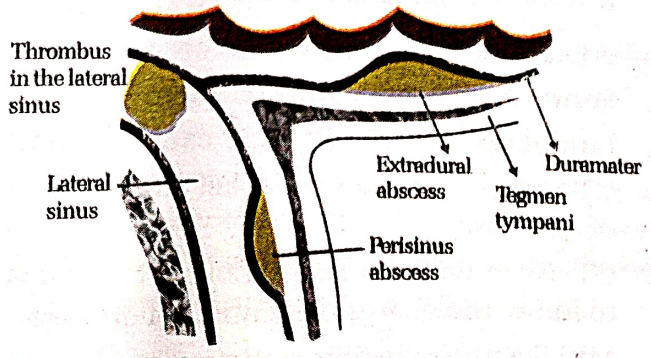
I) Headache on the affected side
ii) Malaise
iii) Chronic ear discharge

H/O relief of headache after ear discharge indicates communication of abscess to the middle ear.

CT scan will confirm the diagnosis and differentiate it from brain abscess.

Treatment

Constitutes a mastoidectomy with drainage of the abscess, following a course of antibiotics.



Meningeal Complications

2. SUBDURAL ABSCESS (Empyema)

Forms when pus tracks into the subdural space. (Non-haemolytic streptococci, Anaerobic Strepto-milleri, Haemophilus influenzae).

Clinical features

Headache and fever are the only symptoms. The findings usually made by chance at operation but may mimic a brain abscess. The patient is investigated for pyrexia of unknown origin and for severe headache occuring without brain damage.

Drowsiness

- Coma, Paralysis of one upper limb – Hemiplegia,
  Hemianopia , hemianaesthesia .
  Aphasia, Jacksonian type of seizures starts at one point and spreads to affect one side of body.

One can determine the site of abscess by the appearance of site of fits and pattern of weakness.

CT and MRI scans can be done to confirm the diagnosis.

(To differentiate it from brain abscess and lateral sinus thrombosis)

Treatment

Drainage of the abscess by a mastoidectomy.
Antibiotics to control infection .

3. LATERAL SINUS THROMBOPHLEBITIS

It is the result of

i. Phlebitis due to contact of the sinus with a perisinus abscess or infected bone. This could be due to an acute or chronic mastoiditis.
ii. Retrograde thrombosis beginning from the mastoid, labyrinth or the tympanic cavity.
iii. Surgical trauma to the sinus.

Clinical features

No symptoms occur unless the organism reaches the blood stream to cause septicemia. Such a thrombosis may be even found unexpectedly at an operation.

Diagnosis

i. ***Temperature*** – Appearing to be normal between two bouts of fever.
(picket - fence pattern)

Rigors are caused by escape of organisms into circulation.

ii. ***Greisinger's sign*** – May be present. A postaural swelling is seen resulting from inflammation of the mastoid emissary vein. (hampers the venous drainage)

iii. ***Headache*** – Due to raised intracranial pressure following venous obstruction.

iv. ***Papilloedema*** – Due to the venous obstruction.

v. ***Tenderness along jugular vein*** – Detected when thrombophlebitis spreads along the vein.

vi. ***Tobey – Ayer test*** – Done by recording the CSF pressure following compression on the jugular veins.

Lillie - Crowe sign.

On occluding jugular vein, opposite to the suspected thrombosed lateral sinus, it produces dilatation of retinal veins on the same side of compression which can be seen on fundoscopy. (Helps to identify which side sinus is involved)

CT Scan shows inflammed sinus and dura i.e. empty triangle (enhancement of walls, not inside of triangle) or " Delta Sign".

(To differentiate it from brain abscess and subdural abscess).

Treatment

1. Antibiotics.
2. Mastoidectomy with adequate exposure of the sinus.
3. Incision and evacuation of the infected clot.
4. Ligation of internal jugular vein if fresh bleeding is not obtained from the jugular bulb.

4. OTITIC HYDROCEPHALUS

(Benign intracranial hypertension)

Occurs frequently in children and adolescents as a complication of acute or chronic otitis media. Always a sinus thrombosis is present which has upset intracranial hemodynamics.

Thrombosis may extend from the sigmoid sinus to the superior sagital sinus leading to reduction in CSF absorption which leads to an increase in CSF pressure.

Clinical features

1. Headache, vomiting and blurring of vision
2. Occasionally diplopia.
3. Blurring of vision.

Diagnosis

1. Papilloedema – Is seen after the onset of intracranial complications.
2. Radiography – of the skull and angiography are normal.
3. Lumbar puncture – Shows a marked increase in pressure but bacteriology and biochemistry of CSF is normal.

Treatment

1. Active medical and surgical treatment is directed against sinus thrombosis.
2. Repeated lumbar punctures are done at 48 hrs. intervals to reduce CSF pressure.
3. Shunt operations may be required.
   The aim of treatment is to reduce CSF pressure to prevent optic atrophy & blindness.

5. MENINGITIS

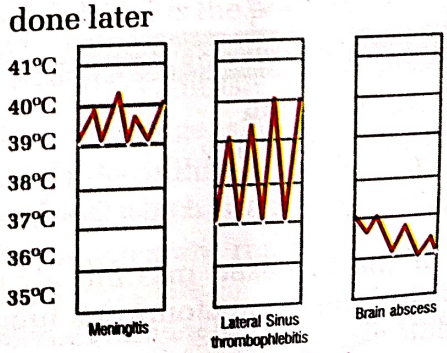
Spread of infection from the tegmen plate, sinus plate, labyrinth or petrous part of temporal bone to the meninges results in meningitis. Depending upon the extent of involvement they are classified as localised or generalised type of meningitis.

Clinical featues

1. Drowsiness
2. Headache, vomiting and fever
3. Neck rigidity with Kernig's and Brudzinski's sign
4. Mental irritability
5. Hemiplegia or cranial nerve palsy

Treatment

1. Antibiotics to control meningitis
2. Cortical or modified Radical Mastoidectomy is done later

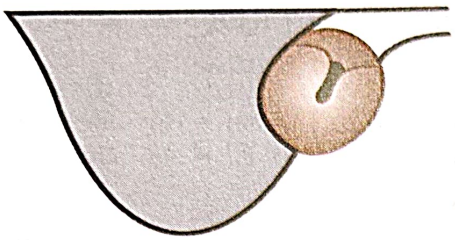


Temperature charts

CHAPTER 11

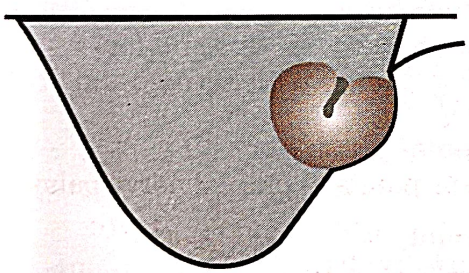
Mastoidectomy

Types :



Simple Mastoidectomy

Radical Mastoidectomy



Modified Radical Mastoidectomy

SIMPLE MASTOIDECTOMY

Schwartze Operation
Cortical mastoidectomy

Definition

Removal of the diseased mastoid air cells without disturbing the anatomy and function of the middle ear.

Indications

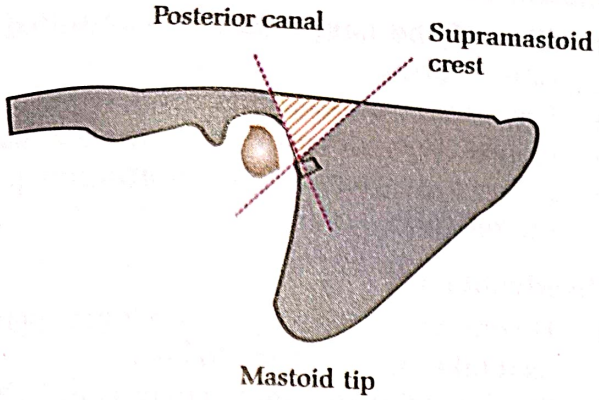
1. Coalescent Mastoiditis
2. Acute otitis media with facial paralysis , not responding to Myringotomy
3. As an approach for
   i. Labyrinthectomy
   ii. Endolymphatic sac decompression
   iii. Facial Nerve decompression
   iv. Vestibulo-Cochlear nerve section
   v. Excision of Acoustic Neuroma
   vi. Petrosectomy

Technique

1. The preliminary steps are the same as that of tympanoplasty.
2. The periosteum over the mastoid is elevated to expose the mastoid bone.
3. The antrum is first located as it is the most consistent and largest mastoid air cell. The landmark to the antrum is the Macewan's or **Supra-meatal triangle.**

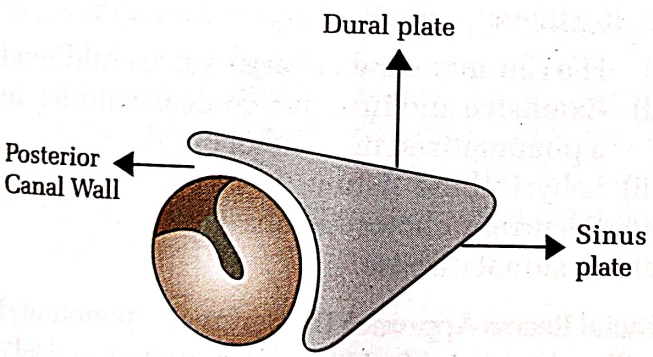
   The boundaries of this triangle are
   i. Superiorly the supramastoid crest.
   ii. Antero-inferiorly the posterior margin of the external canal which cuts the supra-meatal crest.
   iii. Postero-inferiorly a tangent to the external canal which cuts the supra-meatal crest.

   In an adult the mastoid antrum lies 1.5 cm deep to the supra-meatal triangle . The Spine of Henle is useful as :
   i. Landmark for locating the supra-meatal triangle
   ii. Autograft for ossiculoplasty
   iii. For occluding the Eustachian tube opening after a Radical mastoidectomy.



Macewan's Triangle

4. The mastoid antrum is gradually widened with a drill till the following structures are reached :
   i. *Anteriorly* – Posterior canal wall
   ii. *Posteriorly* – Sinus plate
   iii. *Superiorly* – Tegmen plate
   iv. *Inferiorly* – Digastric ridge
   v. *Medially* – Lateral semicircular canal
5. The periosteum is reflected back and closed with catgut.
6. The subcutaneous tissue and skin are sutured to place.
7. Firm mastoid dressing is given.



Limits of a Simple Mastoidectomy

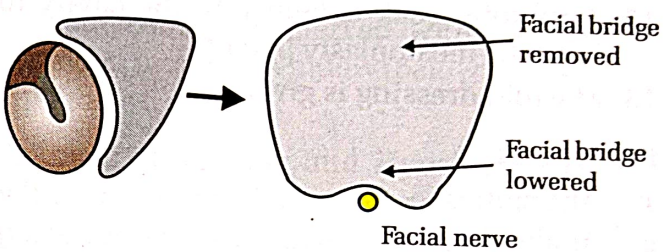
Complications

1. Of Anaesthesia
2. Damage to neighbouring structures
   i. Incus
   ii. Dural plate
   iii. Sigmoid Sinus
   iv. Facial Nerve
   v. Lateral Semicircular canal
3. Perichondritis.

RADICAL MASTOIDECTOMY

Definition:

Eradication of disease from the middle ear cavity, aditus, antrum, with other mastoid air cells and conversion of all these into a single cavity which is exteriorized through the external auditory canal. Here the ossicles are removed and eustachian tube is plugged to separate the middle ear cleft from the nasopharynx.



Simple Mastoidectomy **Radical Mastoidectomy**

Indications

1. Extensive cholesteatoma involving the middle ear, antrum and mastoid air cells with severe sensorineural hearing loss.
2. Cholesteaoma with intracranial complications.
3. Early malignancy of the middle ear cleft.
4. Chronic osteomyelitis of Temporal bone.
5. Tuberculous mastoiditis.

Technique

1. Patient lies supine with the ear to be operated facing upwards.
2. General anaesthesia is preferred.
3. Parts painted, prepared and draped.
4. Steps are similar upto a Simple mastoidectomy.
5. Removal of the Facial Bridge.
   Facial Bridge is the postero-superior aspect of the posterior canal wall and it lies lateral to the attic - aditus.
6. The Facial ridge is lowered .
   Facial ridge is the part of the bony posterior canal wall which is overlying the facial nerve.
7. The ossicles are removed excepting the stapes foot plate.
8. Mucosa overlying the promontary is removed.

9. Eustachian tube orifice is closed with a piece of bone such that no nasopharyngeal infection can later cause ear discharge.
10. Meatoplasty is done to increase the dimensions of the meatus to drain the desquamated epithelium of the skin lined radical cavity. It also allows inspection and application of medicaments into the cavity post-operatively.
11. Closure of the wound in layers.
12. Antibiotic pack is kept into the cavity to keep the meatoplasty patent.
13. Mastoid dressing is given.

Post operative

1. Antibiotics
2. Analgesics
3. Anti-histaminics
4. Pack is removed after 1 week along with sutures.

Complications

1. Of Anaesthesia
2. Damage to various structures
   i. Conchal cartilage
   ii. Sigmoid sinus
   iii. Dura
   iv. Facial Nerve
   v. Semi-circular canal

Causes of persistently discharging cavity :

1. Wrong indications for which surgery is done.
2. Inadequate surgery performed , that is some disease left behind.
3. Patient having Petrositis.
4. Specific diseases like Tuberculosis or Allergy which have to be treated.
5. Retained foreign body like a cotton ball.
6. Focus of infection in nose or throat.

A Radical mastoidectomy gives the patient a cavity open to the exterior. The result of giving such a cavity has its advantages and disadvantages.

Advantages

1. It is an easier and quicker surgery to perform.
2. Allows good exposure.
3. Size of the cavity can be controlled by obliteration.
4. Recurrence of disease easily detected.
5. Complete eradication of disease possible.
6. Allows antibiotics to be applied post-operatively.

Disadvantages

1. Discharge from cavity is for a longer period, as it takes greater time to heal.
2. Deafness due to removal of ossicular chain and tympanic membrane.
3. Dizziness occurs on account of the exposed lateral semi-circular canal.
4. Dependence on the specialist for wax removal at regular intervals.

MODIFIED RADICAL MASTOIDECTOMY

This is similar to a radical mastoidectomy excepting that the ossicular chain is not removed, mucosa over the promontary is left intact and eustachian tube patency maintained. The hearing result is good inspite of cholesteatoma removal.

Done for a cholesteatoma which involves the attic and antrum only.

Indications

i) H/o Chronic ear discharge since childhood
ii) Extensive and invasive choleasteatoma in a pneumatized mastoid of child.
iii) Labyrinthine fistula
iv) Recurrent cholesteatoma
v) Residual disease.

Facial Recess Approach (Posterior tympanotomy) – Simple mastoidectomy with tympanoplasty done by preserving posterior canal wall.

Indications

i) Large pneumatized mastoid
ii) Pneumatization extending upto Zygoma

Contraindications

i) Canal wall up procedure
ii) Only hearing ear
iii) Labyrinthine fistula
iv) Chronic disease
v) Poor Eustachian tube

After completing simple mastoidectomy, vertical portion of facial nerve is identified and then the facial recess is opened in between the posterior annulus and facial nerve by preserving small piece of bone superiorly. Incus is removed after dislocating incudostapedial joint and superiorly Facial recess is removed.

Transcanal anterior atticotomy

Indications :

I) Choleasteatoma involving middle ear, ossicular chain and epitympanum.
ii) Poorly pneumatised zygoma with low tegmen.

Endaural incision is taken, ear canal is circumferentially thinned out from ear floor to the anterior tympanosquamous suture line till mastoid air cells are clearly visible. After identifing the middle fossa dura, incudostapedial joint is separated and choleateatoma is cleared. Obliteration of aditus done with free grafts (cartilage or bone or free muscle) followed by tympanoplasty .

Transcanal hypotympanotomy

Indications : Cholesteatoma of middle ear and hypotympanum (cholesteatoma involving pars tensa).

- Mastoid is saucerized, then posterior annular buttress and bone anterior to facial nerve removed.
- Tympanoplasty done after lowering floor upto hypotympanum.

Anterior - Posterior Mastoidectomy

Intact canal wall procedure

Anterior atticotomy and Hypotympanotomy with a transcortical mastoidectomy.

Indications –

i) Cholesteatoma of Middle ear, Hypotympanum, Epitympanum.
ii) Pneumatised mastoid or temporal bone.
iii) Extensive congenital cholesteatoma.

This method is preferred when disease is extended into oval window (below or posterior) and stapes.

Anterior atticotomy and Hypotympanotomy with a transcortical mastoidectomy done. In this posterior tympanic recess and oval and round windows are exposed by removing posterior annular buttress after clearing disease.

Bondy's modified radical mastoidectomy

Indications –

1) Cholesteatoma limited to mastoid.
2) Only or better hearing ear.
3) In suspected cases of Labyrinthine fistula.
4) Poorly developed mastoid.

In this Modified Radical Mastoidectomy done but middle ear space is not entered.

CHAPTER 12

Non-Suppurative Otitis Media

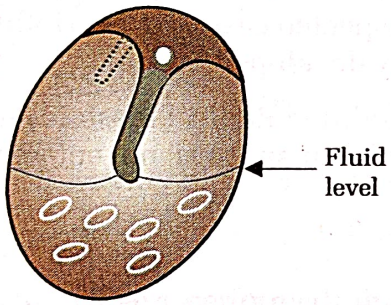
These are conditions which are supposed to be of inflammatory origin but no evidence of suppuration :

1. Serous otitis media
2. Atelectasis
3. Adhesive otitis media
4. Tympanosclerosis

1. SEROUS OTITIS MEDIA

Secretory otitis media
Glue ear
Catarrhal Otitis media
Serous or Non-Suppurative otitis media
Otitis media with effusion

It is characterised by the presence of nonpurulent fluid in the middle ear, also known as 'glue ear'. This fluid may be serous or mucoid, but never purulent.



Serous otitis media

Etiology

Malfunction of the eustachian tube is the underlying cause and the predisposing factors may be :

i. Upper respiratory tract infection
ii. Adenoid and Tonsillar hypertrophy
iii. Tumours of nasopharynx
iv. Otitic barotrauma
v. Radiation therapy
vi. Antibiotic suppressed otitis media
vii. Cleft palate
viii. Systemic diseases like hypothyroidism.

Pathology -

It occurs due to changes in the muco-ciliary mechanism of eustachian tube and middle ear. The mucociliary changes are due to dysfunction of Eustachian tube and nasopharyngeal infection. The conditions affecting normal mucociliary system of Nose and nasopharynx predisposes the formation of fluid, and the negative pressure will cause collection of fluid in the middle ear space.

Clinical features

1. ***Deafness*** – Which is of mild conductive type.
2. ***Fullness*** – With sensation of fluid moving in the ear.
3. ***Air bubbles or fluid*** – May be seen on otoscopic examination. The tympanic membrane may be intact, dull or retracted with restricted mobility.
4. ***Tuning fork test*** – Reveal conductive deafness. A negative Rinne test predict a hearing loss of about 15 to 20 dB.

Investigations

1. ***Pure tone audiometry*** – Shows mild to moderate conductive deafnes.
   It is associated with a mean hearing loss of 20 dB. The test helps in monitoring progress and results of the treatment.
2. ***Impendence Audiometry (Tympanometry)*** – Shows flat type B curve.
3. ***Tympanocentesis*** – Is the gold standard for diagnosis of middle ear fuid.

It is the aspiration of middle ear fluid with spinal needle. Some times it is difficult to aspirate thick glue, then myringotomy should be done.

Treatment

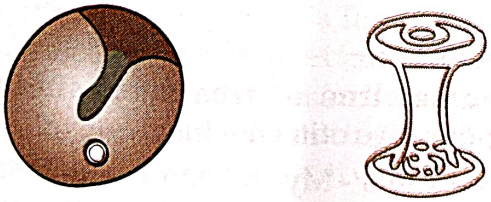
1. *Valsalva's inflation* – to improve eustachian tube function. (Not to be done in rhinitis).
2. *Local decongestant* – nose drops as well as antihistaminics for the same.
3. *In children Adenoidectomy* done in the hope of removal of source of infection.
3. *Myringotomy* – may be required, if no response to medical line of treatment.
4. *Grommet insertion* – when recurrence seen after a myringotomy.

GROMMET INSERTION (Pressure Equalization tube - PET/Tympanotomy tube, Ventilating tubes).

In serous otitis media, even after a myringotomy if there is refilling of the middle ear with fluid, it necessitates a grommet insertion. A grommet is a tube made of an inert material with broadened ends. The purpose of insertion is to establish adequate aeration of the middle ear cleft, with consequent reversion of the pathological changes in the middle ear mucosa.

As such these do not cure the condition but results in temporary improvement in hearing thresholds till they function.

This procedure was first described by Politzer but popularized by Armstrong (devised plastic flanged tube).



Grommet

Rationale of grommet insertion

1. **Drainage of the fluid of middle ear leads to immediate improvement of hearing. It is therefore useful in children.**
2. **To accelerate the return of normal eustachian tube function by aeration of the mucosa. This helps to reverse the pathology of the middle ear mucosa which was caused by prolonged negative middle ear pressure.**
3. **To tide over the problems of adenoid hypertrophy and underdeveloped tensor palati muscles seen in childhood.**

Criteria for an ideal grommet - should

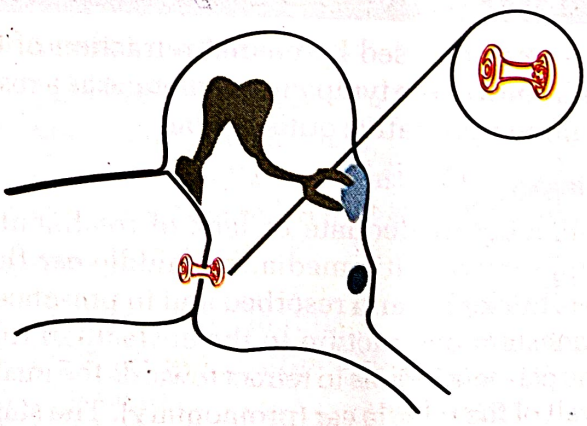
1. Be easy to insert
2. Not get extruded
3. Remain patent
4. Not produce tissue reaction
5. Resist passage
6. Not be expensive

Types of grommets :

Short term grommets	*Long term grommets*
- Shepard tube - Shaw grommet - Reuter bobbin - Armstrong bevelled tube - Paparella type I tube - Feuerstein split tube - Linderman-Silverstein arrow tube	Per-Lee tube Goode T-tube Paparella type 2 and 3 tubes

Technique

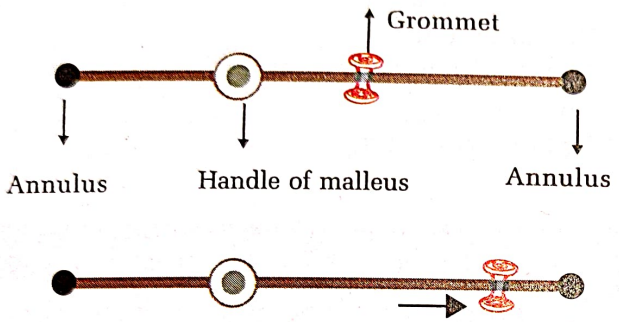
1. General Anaesthesia is used for children only, while local for adults.
2. In the antero-inferior quadrant, an incision is made of the size just smaller than that of the grommet.
3. The grommet is introduced such that , one half of it lies in the middle ear and other half in the canal.



Grommet in place

Complications

1. Anaesthesia.
2. Middle ear infection, as direct communication occurs between the middle and external ear.
3. Perforation of tympanic membrane.
4. Damage to middle ear structures.
5. Obstruction of grommet either by thick secretions, hemorrhage or epithelial debris.
6. Extrusion of the grommet.



Peripheral migration of the grommet

Sequelae

The grommet is gradually pushed towards the annulus on account of peripheral migration of epithelium. The heaped up epithelium under the grommet eventually pushes the tube past the annulus into the external canal. This process takes 6 months to a year.

Some times they may need re-insertion of short term tubes after child had the next attack.

When Grommet does not reverse the middle ear pathology in glue ear, one may require a Cortical Mastoidectomy.

2. ATELECTASIS

It is characterised by medial retraction of the pars tensa of the tympanic membrane as a result of non-suppurative otitis media.

Etiology

Following inadequate or lack of treatment of suppurative otitis media,the middle ear fluid gets thickened and resorbed and in presence of persistant obstruction to the eustachian tube, the pars tensa tends to retract towards the medial wall of the middle ear (promontary). The stages are as follows:

1. The pars tensa is retracted medially but,does not touch the incudostapedial joint.
2. The pars tensa is retracted medially and lies over the incudostapedial joint.
3. The pars tensa lies over the promontory but there are no adhesions between the promontory and the pars tensa.
4. The pars tensa is adherent to the promontary and lies over it,this stage is also known as Adhesive otitis media and may result in cholesteatoma formation with erosion of the ossicular chain.
5. The pars tensa tends to perforate in the anterior or posterior aspect and squamous epithelium is seen draping the promontary,

Clinical Features

1. Deafness - which is moderate to severe conductive type.
2. Tinnitus
3. Otalgia.

Diagnosis

1. Tuning fork tests and Pure tone audiometry reveal moderate to severe degree of conductive hearing loss.
2. Impedance audiometry will show a "Type B" flat curve characteristic of Atelectasis in stages 1 to 4. However this test requires an intact drum and not possible in stage 5 atelectasis.

Treatment

1. Medical line of treatment similar to suppurative otitis media.
2. Stage 1 to 3 - Myringotomy with grommet (Tympano mastoidectomy and ossiculoplasty if hearing loss is more indicating ossicular necrosis).
3. Stage 4 & 5 - Mastoidectomy (Tympanomastoidectomy using sialastic sheets used recently to prevent adhesions).
4. In absence of sequelae,a hearing aid is most effective.

3. ADHESIVE OTITIS MEDIA

It is characterised by fibrous adhesions between the tympanic membrane and the medial wall of middle ear.

4. TYMPANOSCLEROSIS

It is a condition characterised by subepithelial deposition of chalky calcarious plaques in the middle ear or tympanic membrane.

Etiology

There is a hyaline degeneration of the fibrous layer of the middle ear mucosa followed by calcifications which appear like chalky patches in the ear drum or around the ossicular chain.

It is the end stage of chronic inflammatory ear disease. The sites affected are:

1. Tympanic membrane
2. Oval window and stapes foot plate
3. Incudostapedial joint
4. Round window
5. Promontary.

Clinical features

1. Asymptomatic
2. Deafness – Usually conductive,rarely sensorineural if inner ear involved through round or oval window
3. Tinnitus and vertigo if inner ear is involved.

Diagnosis

1. Tuning fork tests and pure tone audiometry reveal conductive hearing loss.
2. Impedance may show a low peaked curve if tympanic membrane is involved or a "Type As" curve if the ossicular chain is fixed due to involvement. If ossicular necrosis has occurred a "Type Ad" curve is seen.
3. White chalky plaques on the tympanic membrane.

Treatment

1. If asymptomatic no treatment is required.
2. If conductive hearing loss,exploratory tympanotomy with plaque removal (onion peel technique) and tympanoplasty is done.
3. If oval or round window involved with sensorineural loss, a hearing aid is required.

ACUTE NECROTISING OTITS MEDIA

This fulminant type of otitis media is seen in acutely febrile, toxic infants and children.

Most commonly Scarlet fever, Influenza, Measles, Pneumonia and other systemic febrile conditions cause necrosis of tympanic membrane, annulus, ossicular chain, mastoid bone and surrounding mucoperiosteum. It is caused by β haemolytic streptococci. These children present with earache, fever followed by mucopurulent foul smelling discharge. They may also present with moderate conductive hearing loss or profound to total sensorineural deafness. Otoscopy reveals small or large central kidney shaped perforation involving pars tensa. The central part always affect first, because if its poor blood supply. In some cases healing occurs by cicatrization of tympanic membrane which appears as thick and atrophic. In severe cases the mastoid antrum with middle ear sloughed by the proteolytic enzymes released by degenerating polymorphonuclear leukocytes. In these cases healing occurs by ingrowth of mucosa (or squamous epithelium) which extend to the epitympanum and antrum (cholesteatoma).

Investigations

i) Complete blood count, ESR
ii) Random Blood sugar
iii) Pure Tone Audiometry is not possible in children, so
iv) Impedence audiometry or BERA preferred
v) X-Ray Mastoid - Schuller's view
vi) CT Scan.

Treatment

i) Systemic antibiotics
ii) Microscopic Aural toilet
iii) In some cases Radical Mastoidectomy done to clear all necrosed and sloughed tissue.

Complications

i) Coalescent mastoiditis
ii) Sigmoid sinus thrombophlebitis
iii) Meningitis
iv) Cerebellar Brain abscess.

CHAPTER 13
Earache

Causes of earache :

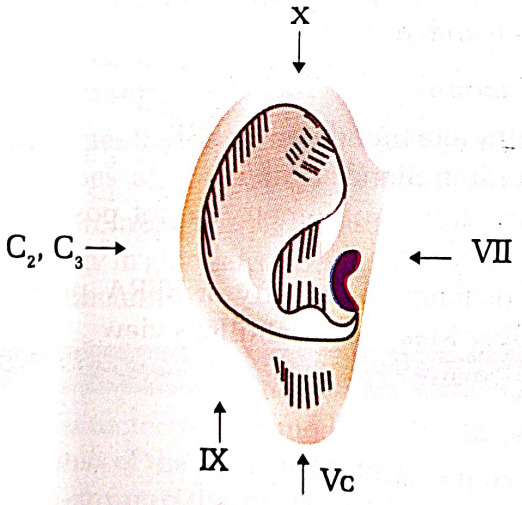
I. LOCAL

A) *External ear*

i. Wax
ii. Fungus
iii. Otitis externa
iv. Myringitis
v. Foreign body
vi. Trauma
vii. Tumours (rare)

B) *Middle ear*

i. Eustachian tube block
ii. Acute otitis media
iii. Mastoiditis
iv. Trauma
v. Barotrauma
vi. Complications of otitis media
vii. Tumours



Nerves leading to referred earache

II. REFERRED

These occur due to other painful conditions occuring at other sites but causing earache as the nerve supplying them, also supplies the ear. The causes are considered on their anatomical basis.

A) Via Trigeminal nerve (V):

i. *Teeth*
   a) Impacted wisdom tooth
   b) Caries tooth
   c) Alveolar abscess
   d) Malocclusion (Costen's syndrome)

ii. *Temporo - Mandibular joint*
   a) Arthritis
   b) Clicking joint

iii. *Nose and paranasal sinuses*
   a) Deviated nasal septum
   b) Sinusitis
   c) Malignancy

iv. *Oral cavity*
   a) Traumatic ulcers
   b) Salivary calculus
   c) Infection
   d) Malignancy

v. *Nasopharynx*
   a) Post-adenoidectomy
   b) Nasopharyngitis
   c) Malignancy of nasopharynx

vi. *Trigeminal neuralgia*

B) Via Facial nerve (VII) :

Herpes Zoster Oticus

C) Via Glossopharyngeal nerve (IX) :

i. *Oropharynx*
   a) Tonsillitis
   b) Pharyngitis
   c) Post-tonsillectomy
   d) Quinsy
   e) Retropharyngeal abscess
   f) Parapharyngeal abscess
   g) Malignancy

ii. *Tongue*
   a) Ulceration
   b) Neoplasms

iii. *Elongated styloid process*

iv. *Glossopharyngeal neuralgia*

D) Via Vagus nerve (X) :

Laryngopharynx
a) Trauma
b) Inflammation
c) Malignancy

E) Via Cervical nerves (C_2 C_3) :

a) Spondylosis
b) Myositis
c) Fibrositis
d) Herpetic lesions

Common causes of referred earache

1. Impacted wisdom tooth
2. Pharyngitis and laryngitis
3. Cervical spondylosis
4. Post tonsillectomy - adenoidectomy
5. Temporo - mandibular joint arthritis.

HERPES ZOSTER OTICUS

Ramsat Hunt described the syndrome consisting of facial palsy, hearing loss with lesions on the ear canal.

Clinical features

Symptoms :

1) Severe pain around the ear (Usually appears before vesicles)
2) Deafness (in severe cases- Sensorineural deafness).

Signs : Vesicles around pinna.

Treatment :

1) Tab. Acyclovir 800 mg 5 times a day
2) Strong Analgesics like Narcotics (Morphine) to relieve pain.

Post herpetic neuralgia : In this syndrome pain lasts for more than one month after the appearance of vesicles. It is due to atrophy of the dorsal horn from T4 - T8 and cell loss and fibrosis of T8 ganglion.

Clinical features

Symptoms :

1) Severe pain around ear
2) Ipsilateral facial palsy
3) Senorineural hearing loss

Treatment :

1) Ethyl chloride spray
2) Electrical stimulation
3) Tab. Amytriptyline
4) Vibratory stimulation

GLOSSOPHARYNGEAL NEURALGIA: Harris described the condition, where continuous pain appears in tonsil, root of tongue and ear which lasts from minutes to hours. This pain radiates around ear and neck and is precipitated by swallowing.

Diagnosed by the relief of pain after anaesthezing the pharynx by local anaesthesia, often is the trigger point of pain. Sometimes it is associated with asystole, syncope and convulsions (may be due to Vagus nerve).

Treatment :

Tab. Carbamazepine 100 mg b.d. to 200 mg q.d.s.

Surgery:

1. Section and avulsion of Glossopharyngeal nerve in the neck.
2. Section of Glossopharyngeal nerve in the posterior cranial fossa.
3. Division of upper root of the Vagus nerve.
4. Decompression of Glossopharyngeal nerve from tortuous vertebral or posterior inferior cerebellar vessels.

TRIGEMINAL NEURALGIA

The syndrome is characterised by frequent paroxysms of lancinating pain on the same side of face including auricle, meatus and tragus. Triggerred by movements or touching the face.

Treatment - Carbamazepine 200mg qds.

Alcohol injection at the nerve or sectioning of the nerve or thermocoagulation of the ganglion. These procedures itself cause distressing pain similar to the original condition (anaesthesia dolorosa).

PERIODIC MIGRAINOUS NEURALGIA

(Cluster headache, Horton's syndrome, Alarm clock neuralgia).

The severe pain arising from nose or cheek which spread to ear or temple associated with nose blockage and ipsilateral red eye, which lasts for 30 minutes to 3 hours constitutes the Cluster headache. Patient exhibit typical repetitive movements.

These are treated by the ergot, pizotifen, methysergide or lithium.

CHAPTER 14

Tinnitus

Definition

Sensation of sound in the absence of relevant external acoustic stimulus.

The word tinnitus is coined from Latin word "tinnire" which means "to ring".

Differential diagnosis

1. Auditory hallucinations - are more complex sounds like music, speech etc. that are actually psychic disturbances.
2. Autophony - where the patient hears his own voice.
3. Respiratory sounds.

Causes -

I. *Objective tinnitus*
   1. Vascular
      a) Aneurysms
      b) A-V malformations
   2. Mechanical
      a) Temporo - mandibular arthritis
      b) Patulous eustachian tube
   3. Muscular
      a) Palatal myoclonus
      b) Stapedius / Tensor tympani contractions

II. *Subjective tinnitus*
   1. Meniere's disease
   2. Acoustic neuroma
   3. Hypertension
   4. Diabetes
   5. Labyrinthitis
   6. Ateriosclerosis
   7. Migrane
   8. Epilepsy

Common causes

1. Drugs - salicylates & streptomycin
2. Hypertension, leukemia
3. Migrane
4. Uremia
5. Epilepsy
6. Aortic regurgitation
7. Otologic

Classified according to the site

1. **External ear**
   1. Impacted wax
   2. Polyps
   3. Foreign body.
3. **Middle ear**
   1. Acute otitis media
   2. Serous otitis media
   3. Eustachian tube block.
   4. A-V malformations
4. **Inner ear**
   1. Meniere's disease
   2. Acoustic neuroma
5. **Labyrinthitis**

   Systemic
   - Anaemia
   - Leukemia
   - Hypertension

Treatment

1) Treat the underlying cause, which relieve the tinnitus.
2) Hearing aids
3) Sedatives and anxiolytics
4) Vasodilators like Nylidrine given.
5) Cocaine, Vit. A, Ergot, Dimethyl sulphexide, Nicotinic acid etc.
6) Injections are given when all forms of treatment are ineffective.
   Lignocaine -100mg IV given rapidly.
7) Cortisone -100mg
8) Tinnitus maskers
9) Tympanic neurectomy.

TYMPANIC NEURECTOMY

Definition

It is destruction of the nerve plexus situated on the medial wall of the middle ear.

Constituents of the Nerve plexus

1. Tympanic branch of the Glossopharyngeal nerve.
2. Superior and inferior Carotico-tympanic branches of sympathetic plexus of internal carotid artery.
3. This plexus itself gives a branch to the greater and lesser superficial petrosal nerve.
4. A twig from Facial nerve.

Indications:

1. Drooling of saliva
2. Tympanic neuralgia
3. Crocodile Tears
4. Diffuse parotid enlargement
5. Parotid fistulae
6. Tinnitus
7. Frey's syndrome

Technique

1. Local anaesthesia is preferred, excepting in children.
2. Endomeatal incision is taken, like in a stapedectomy.
3. The middle ear is exposed after the tympanomeatal flap elevation.
4. Nerve plexus situated on the promontary (medial wall of middle ear) sectioned with a sharp instrument(sickle knife).
5. The tympanomeatal flap is reposited back to postion and gelfoam packed into the canal.

Complications

1. Anaesthesia
2. Tympanic membrane tear leading to a perforation
3. Ossicular disruption
4. Facial nerve damage
5. Round window damage
6. Recurrence of symptoms due to regeneration of fibres or incompelete transection.

CHAPTER 15

Otoscleraosis

Definition

Otosclerosis is a localised disease of the bone derived from the otic capsule, where mature lamellar bone is removed by osteoclasis and replaced by unorganised bone of greater thickness, cellularity, and vascularity.

The more appropriate pathological term is '*Otospongiosis*'.

Etiology

1. *Age* – The peak incidence is seen in the third decade.
2. *Sex* – Commoner amongst females , the ratio being 2:1.
3. *Pregnancy* – Causes an initiation or progression of the disease.
4. *Familial* – Predilection of otosclerosis has been recognised in half the patients.
5. *Osteogenesis Imperfecta* – The triad with otosclerosis and blue sclera is called Vander Hoeve syndrome.

   However, the exact cause of otosclerosis is still not known.

Types

1. *Stapedial otosclerosis* – Occurs due to fixation of the footplate of the stapes resulting into a conductive hearing loss.
2. *Cochlear otosclerosis* – Where the new bone formation involves the bony capsule of the labyrinth causing sensorineural hearing loss.
3. *Combined otosclerosis* – Results in a mixed type of hearing loss.

Clinical features

The Tympanic membrane is normal on otoscopic examination and nose-throat examination is also normal. Sometimes a reddish glow is seen through an intact tympanic membrane, called Schwartz sign. Depending upon the type of otoslerosis, the tuning fork test and audiometry give results accordingly.

Differential diagnosis

1. Serous otitis media
2. Ossicular dislocation
3. Osteogenesis imperfecta
4. Paget's disease.

Treatment

A) Medical treatment is advised for
   i. Cochlear otosclerosis
   ii. Combined otosclerosis
   iii. Stapedial otosclerosis when surgery is contraindicated.

   20-60 mg sodium fluoride is given daily. A regular renal function test & X-ray chest is done during course of treatment to check for fluorosis.

B) Surgical treatment is stapedectomy.

C) Hearing Aid – Can be used if patient refuses or is not fit for surgery.

STAPEDECTOMY

Definition

It is replacement of the fixed stapes by a prosthesis to convert it into a mobile unit. Performed for stapedial otosclerosis only.

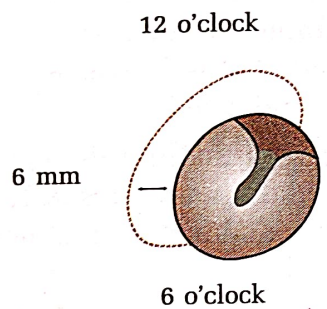
Contraindications

1. Very old patients
2. Children
3. Generalised Medical disorders
4. Poor Eustachian tube function
5. Otitis externa or furuncle in the canal
6. During pregnancy
7. Pagets disease
8. Patients having tinnitus and vertigo

9. Cochlear otosclerosis
10. Other ossicular pathologies e.g. fixed malleus
11. Occupations involving physical strain
12. Unilateral otosclerosis
13. Stapedectomy on the second ear
14. Tympanic membrane perforation

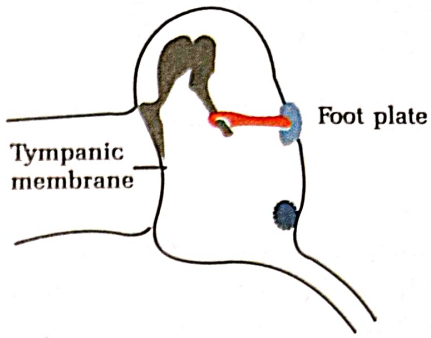
Technique

1. Done under local anaesthesia , as bleeding is minimal and hearing can be tested on table.
2. Incision is made on the canal skin from 6 o'clock to 12 o'clock just 6mm away from the annulus.
3. Canal wall flap elevated and middle ear exposed.



Incision for a stapedectomy (Endomeatal)

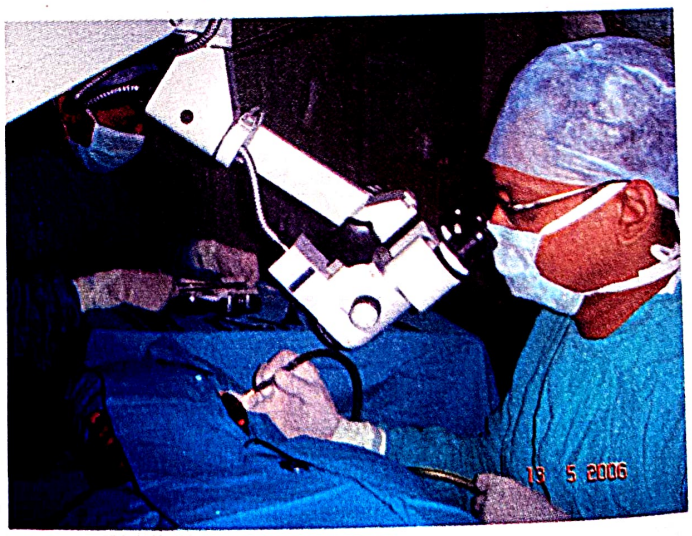
4. The stapedius tendon is cut and opening made in the foot plate of stapes .
5. Stapes super-structure is removed.
6. A Teflon piston is placed such that its tip lies into the opening created in the foot plate of stapes and the other end around the Incus.
7. Hearing is tested on the table.
8. Tympano-meatal flap is reposited back and gelfoam placed in the canal.
9. No sutures are required and cotton plug is placed in the canal.



Teflon Piston (red) in place

Complications

1. Local Anaesthesia.
2. Labyrinthitis due to infection passing to the inner ear via the opening created on the foot plate.
3. Giddiness and vomiting due to inner ear fluids disturbed by a long teflon piston.
4. Dislocation of piston from Incus leading to a conductive hearing loss.
5. Facial Nerve paralysis.
6. Perforation due to tympanic membrane damage.
7. Alteration of taste sensation due to chorda tympani being stretched or damaged.
8. Sensorineural hearing loss due to surgical trauma or a perilymph fistula.



Stapedectomy via endomeatal incision

CHAPTER 16

Otorrhoea

Otorrhoea is ear discharge. It is due to conditions affecting the ear or sometimes the neighbouring structures around the ear.

CAUSES

A) In the Ear :

1. ***External ear***
   - i. Acute otitis externa
   - ii. Otomycosis
   - iii. Burst furuncle
   - iv. Myringitis
   - v. Eczematous otitis externa
   - vi. Seborrhoeic otitis externa
   - vii. Tumours : Ceruminoma, etc
   - viii. Malignant tumours
   - ix. Wax with secondary otitis externa
   - x. Granulomas
   - xi. Viral otitis externa
2. ***Middle ear***
   - i. Acute suppurative otitis media
   - ii. Chronic suppurative otitis media
   - iii. Tumours (rare)
3. ***Inner ear***
   Suppurative Labyrinthitis leading to perilymph fistula adding to otorrhoea caused by otitis media

B) Outside the Ear :

- i. CSF otorrhea
- ii. Parotid abscess rupturing into external auditory canal
- iii. Temporo-mandibular joint abscess rupturing into external auditory canal.

Types of discharge :

1. ***Serous*** – Eczematous otitis externa
2. ***Mucoid*** – Central perforation (safe type)
3. ***Purulent*** – Burst furuncle, abscess from the parotid or temporo-mandibular joint and malignant otitis externa.
4. ***Foul smelling*** – Attic perforation (unsafe type)
5. ***Blood stained*** – Polyp, granulations, trauma and tumours.
6. ***Watery*** – CSF otorrhea and myringitis.

Investigations

1. Ear examination
2. Ear discharge sent for smear, culture and antibiotic sensitivity (SCABS)
3. Exfoliative cytology
4. Hearing tests
5. X-ray mastoid
6. Biopsy to exclude malignancy.

Management:

1. ***General*** –
   - a) Aural toilet with suction or swab sticks
   - b) Prevention of water entering the ear
   - c) Systemic antibiotics if required
   - d) Ear drops, especially steroids or antibiotics (contraindicated in fungal infections and in CSF otorrhoea).
2. ***Specific*** – Depending on the cause.

CHAPTER 17

Middle Ear Injuries

Types of Middle Ear injuries :

1) Otitic barotrauma
2) Traumatic rupture of the tympanic membrane
3) Ossicular discontinuity
4) Haemotympanum.

OTITIC BAROTRAUMA

A non-infective inflammatory reaction in the lining of the middle-ear occurs when air pressure within it, is considerably below that of the surrounding atmospheric pressure i.e. in a relatively negative intratympanic pressure.

Etiology :

The eustachian tube is normally opened by muscular action and this allows pressure within the middle ear to be raised to that of the surrounding atmosphere. The eustachian tube when cannot open up, is said to be 'locked'. This occurs when the difference of pressure outside and that within the middle ear is more than 80 mm of Hg.

Conditions – adenoiditis, sinusitis, rhinitis etc. which cause eustachian tube block lead to such a situation. Similarly rapid descent in an aircraft may produce blocking of the eustachian tube.

Clinical features

i. ***Deafness*** – Develops progressively and relieved by swallowing movement.

ii. ***Pain and discomfort*** – Occurs when the block is severe.

iii. ***Tinnitus*** – May be present .

iv. ***Vertigo*** – When the block is unilateral .

v. ***Otoscopy*** – Shows a red and congested drum, bubbles or fluid may be seen later and in severe cases perforation occurs.

Treatment

1. Autoinflation with swallowing movements or Valsalva's manoeuvre is performed.
2. Decongestants to decrease eustachian tube obstruction.
3. Myringotomy if fluid is present.
4. Treatment of cause.

Prevention

1. Avoidance of flight during an upper respiratory infection.
2. Avoid sleeping during descent in a flight , as eustachian tubes are not opened during sleep.
3. Use of nasal decongestants before a flight.
4. Auto-inflation of the tube.
5. Grommet insertion in recurrent cases.
6. The modified Valsalva manoeuvre (Controlled expiration with lips closed and blocking of nose with fingers) or Frenzel manoeuvre (Simultaneous contraction of floor of mouth muscles with closure of glottis, mouth and nose) should be done.
7. Children with cold should avoid flying. If can't avoid flying they should be encouraged to swallow repeatedly or chewgum. To prevent this in neonates they should be bottle fed.

High risk individuals for aero-otitis media-

1) H/o Otitis media
2) Inflammatory disease of mastoid or middle ear.
3) H/o mastoid or middle ear surgery.
4) Patients with anatomical defects, hypertrophied turbinates or nasal polyps can affect middle ear mechanism .

TRAUMATIC RUPTURE OF TYMPANIC MEMBRANE

Tympanic membrane tear results following an injury.

Etiology

1. Slap on the ear.
2. Instrumentation during cleaning the ear.
3. Foreign body, insertion or its attempted removal.
4. Valsalva's manoeuvre done forcibly.
5. Head injury

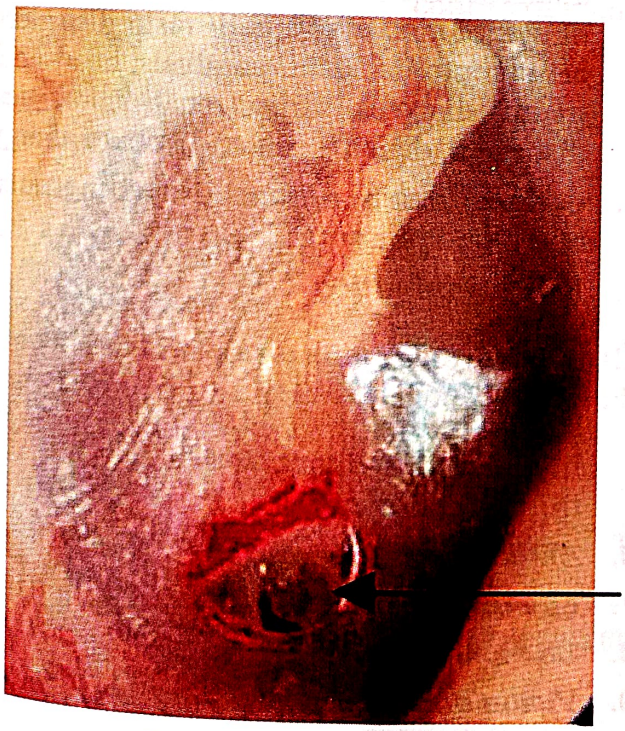
Symptoms

1. Earache.
2. Deafness.
3. Ear bleed.
4. Vertigo.

Irregular shaped perforation is seen & ossicular discontinuity may be present.

Management

1. Conservative - ear drops are avoided in case of injuries.
2. Small sized perforations heal in 3 weeks.
3. Large perforations would require a Myringoplasty or even Tympanoplasty.



Traumatic Perforation

OSSICULAR DISCONTINUITY

Following ossicular discontinuity the patient has conductive deafness. The diagnosis is confirmed on Impedance audiometry which shows a disjoined peak.

Management –A Tympanoplasty is done, depending on the type of ossicular disruption.

HAEMOTYMPANUM

Following trauma or surgery there is a collection of blood in the middle ear.

On examination the tympanic membrane appears blue in colour and patient has a conductive hearing loss.

Frequently haemorrhage is seen in bleeding disorders like leukemia, thrombocytopenic purpura, haemophilia and fracture temporal bone. Postoperative haemotympanum resolves spontaneously or drains out through Eustachian tube in about 10 days without any problems.

Idiopathic hemotympanum (blue ear drum) is a variant of otitis media with effusion, characterized by bluish, or brown or yellow coloured fluid in the pneumatised spaces of the temporal bone. The change in colour is due to haemolysis of blood.

Management

1. Anti-inflammatory & nasal decongestants. The blood gets absorbed in the middle ear or gets drained through the eustachian tube.
2. Myringotomy done, if blood does not resolve from the middle ear.

CHAPTER 18
Vertigo

Vertigo is hallucination of movement. The two vestibular organs work together in a synchronous manner. Each of them has a resting action potential, having an equal and opposite effect upon the eye and the skeletal system for purpose of maintaining equilibrium.

Causes :

I. PERIPHERAL

A) ***External ear***
- i. Wax
- ii. Furunculosis

B) ***Middle ear***
- i. Acute otitis media
- ii. Eustachian tube block

C) ***Inner ear or end organ***
- i. Meniere's disease
- ii. Labyrinthititis
- iii. Vestibular neuropathy
- iv. Benign Positional Paroxysmal Vertigo (BPPV)

II. CENTRAL

V - Vascular
- i. Vertebrobasilar insufficiency
- ii. Migrane
- iii. Stroke
- iv. Vasculitis

E - Epilepsy
- i. Preceding a seizure
- ii. Following a seizure

R - Rx
- i. Aspirin
- ii. Anti-hypertensive drugs
- iii. Tranquilizers
- iv. Quinine

T - Tumour
- i. Acoustic neuroma
- ii. Gliomas

Trauma
- i. Head injury
- ii. Whiplash injury

Thyroid
Hypothyroidism

I - Infections
- i. Viral encephalitis
- ii. Bacterial meningitis
- iii. Syphillis
- iv. Vestibular neuronitis

G - Glial diseases
Multiple sclerosis

O - Ocular pathology
- i. Dioptic abnormalities
- ii. Looking down from heights
- iii. Extra-ocular muscles weakness causing diplopia.

Investigations:

1. ***Detailed History*** – Regarding the type of attack of vertigo, triggering factors, duration of symptoms, eye/ smell/ taste disorders, ear symptoms, facial paralysis, head-neck trauma, diabetes, cardiac/ renal/ neurological disorders, drugs taken and known allergy.
2. ***General examination*** – Including pulse and blood pressure.
3. ***ENT examination***
4. ***Blood tests***
5. ***Radiology***
   - i. Mastoid
   - ii. Internal auditory meatus
   - iii. Cervical spine
6. ***Audiometry***
7. ***Electrocardiogram***
8. ***CSF examination***
9. ***Vision and fundus examination***
10. ***CT Scan.***

	Peripheral nystagmus	Central nystagmus
a) Incidence	90% of cases	10% of cases
b) Latency	2-10 sec	More than 1 minute
c) Duration	Stops within 30 sec.	None
d) Adaption	Disappears in 50 sec.	Persists
e) Fatiguabiliy	Disappears on repetition	Repeatable
f) Direction	Only to one side	Changes with change of position
g) Position	Present only in one side	Present in multiple positions
h) Vertigo	Always present and severe	Absent or mild

Management

A. *Medical :*

1. ***Reassurance*** – Regarding the nature of the disease.
2. ***Labyrinthine sedatives*** – Are given to control the nausea ,vomiting and giddiness e.g. prochloroperazine (stemetil) or dimenhydrinate (dramamine).
3. ***Diuretics*** – With a low salt diet is advised to reduce endolymphatic hydrops e.g. furosemide.
4. ***Tranquillizers and sedatives*** – Given to reduce anxiety by suppresing the activity of medial vestibular nucleus e.g. calmpose.
5. ***Antihistamines*** – Are given for their vasodilator action e.g. cinnarizine or betahistidine.
6. ***Histamine drip*** – Helps to control the acute attacks.

B. *General :*

1. ***Septic foci*** – Are treated.
2. ***Smoking*** – To be stopped.
3. ***Alcohol*** – Consumption reduced.

C. *Labyrinthine exercises :*

Cawthorne-Cooksey exercises are taught to the patient to achieve habituation and also help in regaining confidence.

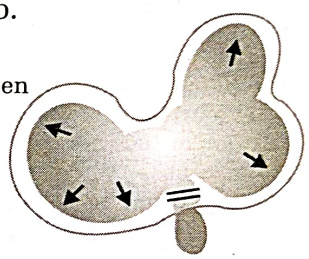
D. *Surgery :*

1. ***Endolymphatic sac decompression*** – To reduce the tension of endolymph .
2. ***Destruction of vestibule*** – By cryosurgery or ultrasound.
3. Vestibular nerve section.
4. ***Labyrinthectomy*** – Results in complete hearing loss in the affected ear.

MENIERE'S DISEASE

Definition

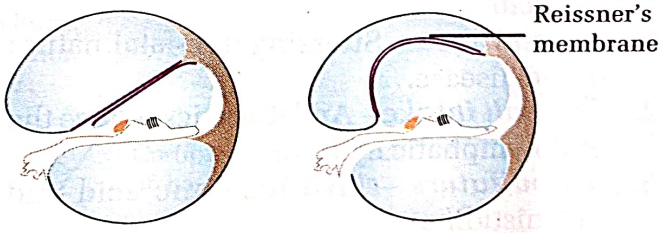
Meniere's disease is idiopathic endolymphatic hydrops characterised by fluctuating hearing loss, tinnitus and vertigo.



Endolymphatic hydrops. Drainage via the sacus has been impeded and the membranous labyrinth has become distended with endolymph

Etiology

1. ***Age*** – Occurs before the age of 50 years.
2. ***Sex*** – Commoner in males.
3. ***Unilateral*** – Involvement is seen more often than bilateral.
4. ***Idiopathic.***



Normal Meniere's syndrome

Endolymphatic compartment

Pathology

1. There is distension of the endolymphatic spaces affecting mainly the cochlea and saccule.
2. May be associated with rupture of the membranous labyrinth.

Clinical features :

1. ***Fluctuating hearing loss*** – Later becoming a permanent sensorineural hearing loss.
2. ***Roaring tinnitus*** – Is the most prominent symptom before an attack.
3. ***Vertigo*** – May range from dizziness to severe giddiness.
4. ***Vagal signs*** – Like pallor, sweating and slowing of pulse.
5. ***Sensation of fullness*** – In the ear.
6. ***Anxiety state***

Investigations:

1. ***Otoscopy*** – Shows a normal tympanic membrane.
2. ***Hearing tests*** – Reveal a sensorineural loss.
3. ***Caloric test*** – Gives variable results.
4. ***Glycerol test*** – Is diagnostic of Meniere's disease.
5. ***Electronystagmography.***
6. Other tests as performed for vertigo patients.

Differential diagnosis

	Meniere's Disease	Vestibular neuronitis	Positional vertigo	Acoustic neuroma
(a) Giddiness	Irregular, recurrent.	Acute onset, recurrence uncommon	Recurrent, momentary	Chronic unsteadiness
(b) Nausea and vomiting.	Present	Present	Absent	Absent, Present in late stage.
(c) Sensorineural deafness	Present, Fluctuating	Absent	Absent	Present, Progressive.
(d) Recruitment	Present	Absent	Absent	Absent
(e) Tinnitus	Present	Absent	Absent	Present
(f) Cranial nerve palsies	Absent	Absent	May be present with central type	May be present

Treatment:

I. *General*

1. Smoking and alcohol to be discontinued.
2. Septic focus is treated.

II. *Medical*

1. ***Reassurance*** – Stressing non-fatal nature of the disease.
2. ***Low salt intake*** – And diuretics reduce the endolymphatic hydrops.
3. ***Vasodilators*** – Like nicotinic acid and betahistidine.
4. ***Labyrinthine sedatives*** – Like dimenhydrinate and prochlorperazine.
5. ***Antihistaminics*** – Like cinnarazine for vestibular suppressant activity.
6. ***Tranquillizers*** – To allay anxiety.

III. Surgical

Types of Surgery

1. To reduce endolymph production - Sympathectomy.
2. To increase the endolymph absorption - Endolymphatic sac decompression.
3. Relief of symptoms by denervation of the vestibular labyrinth – Vestibular neurectomy.
4. Destruction of the Labyrinth -Labyrinthectomy.

Endolymphatic sac decompression is preferred as

i. It reduces the tinnitus.
ii. Giddiness is treated.
iii. Hearing is not hampered.

ACOUSTIC NEUROMA

Also referred as Vestibular Schwannoma or Eight nerve tumour.

Etiopathology

The tumour arises from the schwann cells of the vestibular nerve within the internal auditory canal. It is a slow growing tumour gradually widening and eroding the canal, later entering the cerebropontine angle.

Clinical features

1. ***Age :*** Seen after the age of 40 years.
2. ***Sex :*** Commoner in females than males.
3. ***Vestibulo-cochlear :*** Occur the earliest even when the tumour is intra-canalicular. The classical symptom being a progressive unilateral sensori neural hearing loss associated with tinnitus.
4. ***Cranial nerve :*** The Trigemminal & Facial nerve get involved first and later the Glossopharyngeal and Vagus nerve get affected leading to dysphagia and hoarseness of voice.
5. ***Brainstem :*** involvement leads to weakness of the extremities and exaggerated reflexes.
6. ***Cerebellar :*** Involvement occurs only with large tumours.
7. ***Raised intracranial tension :*** Too seen with large tumours and patients present with headache, vomiting & blurring of vision.

Investigations

1. ***Audiological***
   a. ***Pure tone audiometry*** – Sensori neural hearing loss.
   b. ***Speech audiometry*** – Poor speech discrimination.
   c. ***Tone decay*** – Retrocochlear lesion.
   d. ***Recruitment phenomenon*** – Absent.
2. ***Vestibular***
   With very small tumour the test would be normal, otherwise the caloric test is absent or diminished.
3. ***Neurological***
   Examination of the cranial nerves, cerebellar signs & other neurological examinations.
4. ***CSF examination***
   Punctures usually not done.
5. ***Evoked response audiometry***
   There is a latency in wave 5 which is diagnostic of the tumour.
6. ***Radiology***
   a) X-rays – Widening of the internal auditory canal
   b) CT scan – Detects small intracanalicular tumours.
   c) MRI scan – Earliest method to diagnose acoustic tumours.

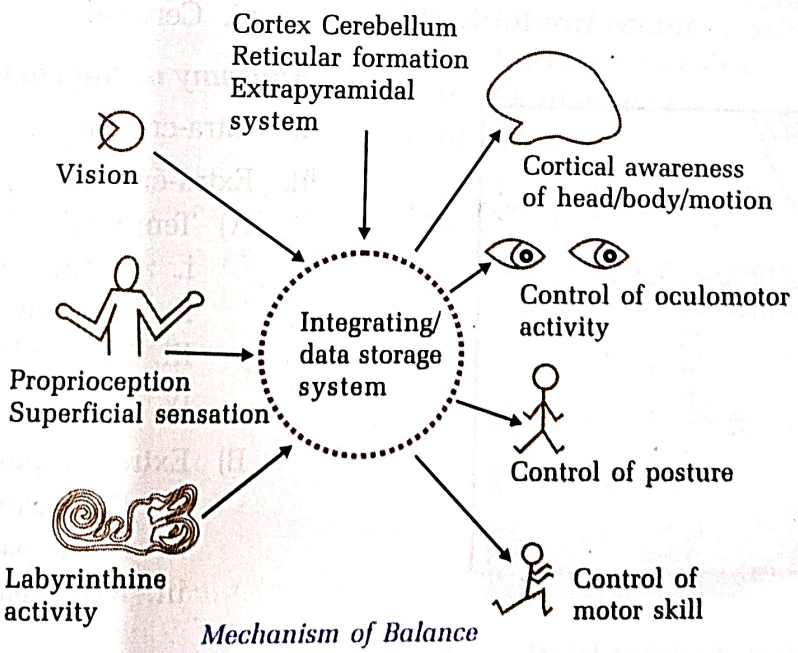
Differential Diagnosis

1. Meniere's disease
2. Cholesteatoma
3. Meningioma
4. Arachnoid cyst.

Management

Surgical removal of the tumour. The approaches are :

1. Translabyrinthine
2. Middle cranial fossa
3. Suboccipital
4. Combined approach.



Mechanism of Balance

CHAPTER 19

Facial Paralysis

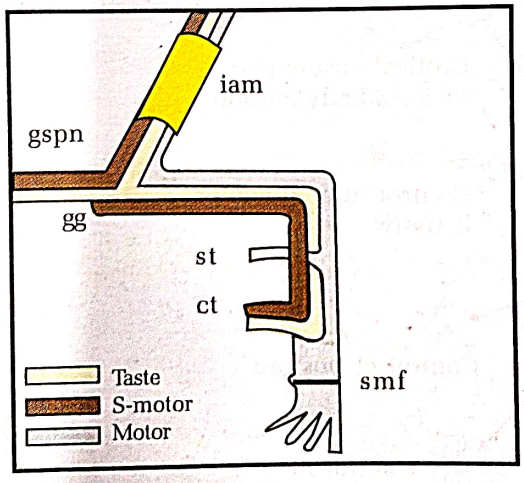
Facial (VII) is the commonest cranial nerve to get affected, which is due to the following reasons :

1. Closely related to important structures at
   i. Cerebello-Pontine Angle
   ii. Vestibulo-Cochlear nerve
   iii. Middle-Ear
   iv. Mastoid
   v. Parotid gland.

Therefore any pathology involving the above structures leads to facial nerve involvement.

2. The Facial Nerve alters its course radically
   i. On exit from the Internal Auditory Canal to form the geniculate ganglion (First genu).
   ii. The second right angle bend at the pyramidal portion (Second genu).
   iii. After exit from the stylomastoid foramen, as it hooks anteriorly into the parotid gland.

3. The Facial Nerve is *encased in a bony canal* which is longest, as compared to any other nerve in the body.



Branches:

1. At the geniculate ganglion, the greater superficial petrosal nerve.
2. A twig to the tympanic plexus.
3. Nerve to stapedius from the pyramidal portion.
4. Chorda tympani from the mastoid portion.
5. Just after leaving the stylomastoid foramen, the branches given are to the stylohyoid, posterior belly of the digastric, a postauricular branch to the occipito-frontalis and external auricular muscles.
6. Within the parotid gland it is divided into an upper zygomatico-temporal branch and a lower cervico-facial branch.
7. On exit from the parotid gland, further branching leads to
   i. Temporal
   ii. Upper-zygomatic
   iii. Lower-zygomatic
   iv. Buccal
   v. Mandibular
   vi. Cervical

Anatomy of the Facial Nerve

I. Intra-cranial

II. Extra-cranial:

A) Temporal
   i. Petrous (Internal Auditory Meatus)
   ii. Tympanic (Horizontal)
   iii. Pyramidal (Second Genu)
   iv. Mastoid (Vertical)

B) Extra-Temporal
   i. Pre-parotid
   ii. In the parotid gland
   iii. Extra-parotid

Signs Differentiating Lesions:

Supranuclear	Infranuclear
1. Forehead muscles intact	Total facial palsy
2. Tongue movements impaired	Tongue movements normal
3. Ataxia	No ataxia
4. Associated hemiplegia	No hemiplegia
5. Tone is maintained	Flaccid Tone
6. Reflexes intact	Absent reflexes
7. No muscle	Muscle atrophy
8. No muscle fasciculations	Muscle fasciculations.

In ENT we would be dealing with Infranuclear type of Facial paralysis.

Causes

I. *Congenital*
i. Birth moulding theory
ii. Forceps delivery
iii. Moebius syndrome

II. *Inflammatory*
i. Otitis Media
ii. Mastoiditis
iii. Otitis Externa
iv. Poliomyelitis
v. Syphillis
vi. Leprosy
vii. Mumps
viii. Chicken Pox
ix. Encephalitis
x. Tuberculosis
xi. Tetanus
xii. Diphtheria

III. *Traumatic*
i. Facial injuries
ii. Base skull fracture
iii. Injury to middle ear

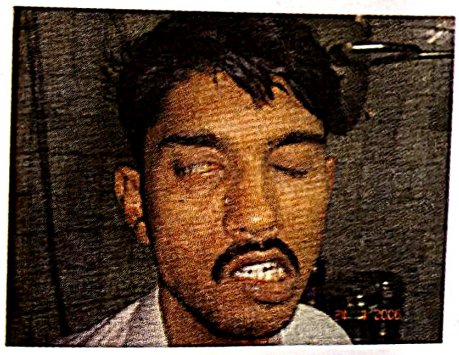
IV. *Metabolic*
i. Pregnancy
ii. Hypertension
iii. Hyperthyroidism
iv. Diabetes
v. Acute Porphyria

V. *Neoplastic*
i. Cholesteatoma
ii. Leukemia
iii. Meningioma
iv. Acoustic Neuroma
v. Glomus Jugulare
vi. Facial Nerve Tumour

VI. *Iatrogenic*
i. Mastoidectomy
ii. Parotidectomy
iii. Embolisation
iv. Post-immunisation

VII. *Idiopathic*
i. Bell's Palsy
ii. Myasthenia Gravis
iii. Osteopetrosis
iv. Polyarteritis nodosa
v. Sarcoidosis

VIII. *Bilateral Facial Paralysis*
i. Infectious mononucleosis
ii. Skull trauma
iii. Leprosy
iv. Acute porphyria
v. Moebius syndrome.



Facial paralysis (right side)

Common causes

i. Bell's palsy
ii. Cholesteatoma
iii. Otitis media
iv. Skull-base fracture
v. Iatrogenic.

BELL'S PALSY

Commonest cause of facial palsy.

Etiology

1. **Age** – found at any age.
2. **Sex** – incidence is higher amongst females, as seen more often during pregnancy.
3. ***Viral infection.***
4. ***Vasospasm***
5. ***Allergy***

Pathology

The most widely accepted theory is that of vasospasm, causing tissue damage within the fallopian canal (primary ischemia). The resulting swelling of the soft tissues within this narrow space with rigid walls causes secondary ischemia and thus neural dysfunction.

Clinical features

1. H/o Exposure to cold
2. Pain in or around the ear
3. Facial asymmetry
4. Loss of taste sensation
5. Drying of the eye
6. Hyperacusis.

Investigations

1. *Laboratory Test:*

i.	Complete blood count	– Infectious mononucleosis and leukemia
ii.	Erythrocyte sedimentation rate	– Sarcoidosis, tuberculosis and collagen disorders
iii.	Urine and stool examination	– Acute porphyria and sarcoidosis
iv.	Blood sugar	– Diabetes mellitus
v.	Marrow examination	– Leukemia and lymphoma
vi.	Cerebrospinal fluid	– Meningitis
vii.	Serum antibody test	– Rheumatoid arthritis and polyarteritis nodosa.

2. ***Radiological:***

i. X-ray for visualising Mastoids and Internal auditory canal
ii. Tomography of the temporal bone.
iii. C.T. Scan
iv. MRI.

3. ***Electrical Tests:***

i. Electro Myography (EMG)
ii. Nerve Excitation Test (NET)
iii. Electro Neuronography (ENG)

4. ***Audiometry:***

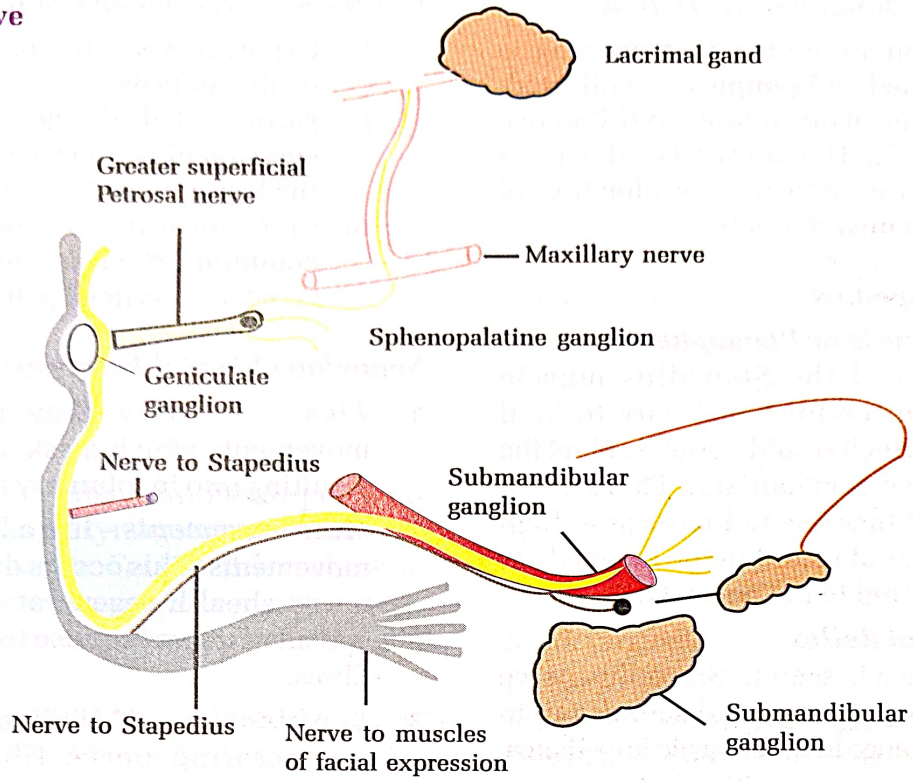
The diagnosis of Facial Paralysis is on the face of the patient. It is important to know the level of lesion of Facial Paralysis.

The following tests are performed and the integrity of the nerve at that particular branch level can be ascertained.

The Facial Nerve

1. Greater superficial Petrosal nerve
2. Nerve to Stapedius
3. Chorda tympani nerve
4. Nerves to muscles of facial expression
5. Geniculate ganglion
6. Sphenopalatine ganglion
7. Submandibular ganglion
8. Maxillary nerve
9. Lacrimal gand
10. Submandibular gland

The Facial Nerve



1. *Tests for Muscles of Facial Expression*

i.	Frontalis	–	Frowning of the forehead muscles
ii.	Orbicularis Oculi	–	Closing the eyes
iii.	Buccinator	–	Blowing the cheeks
iv.	Orbicularis Oris	–	Movement at angle of the mouth
v.	Platysma	–	Neck muscle contraction

Passive Signs (Asymmetry)

i. Palpebral border
ii. Nasolabial fold
iii. Vermillion border (red line of the lip)

The Muscles which are not tested

i. Posterior belly of the diagastric
ii. Stylohoid and
iii. Post-auricular muscles

2. *Testing for Chorda tympani*

i. ***Taste :***

a) Patient is asked to gargle before the test
b) Tongue is pulled out with the help of a gauze piece.
c) Sweet, salt, bitter and sour substances are applied at anterior two-thirds on the half of the tongue without the patient seeing the substances used.
d) If the taste sensation is absent, the test is repeated on the normal side. Unfortunately the identification of these substances becomes difficult in elderly patients and smokers.

ii. ***Electrogustometry***

Quantitative assesment is done to evaluate the amount of current required to elicit an acidic sensation on the antero-lateral border of the tongue. If a current of more than 100 amp is required, then it is concluded that the taste sensation has been lost.

iii. ***Submandibular salivary flow***

The submandibular duct opening is cannulated and samples are collected, each being of one minute. A difference of 25% in the quantity of saliva collected is indicative of affection of chorda tympani function.

3. ***Test for Stapedius***

i. ***Hyperacusis or Phonophobia***

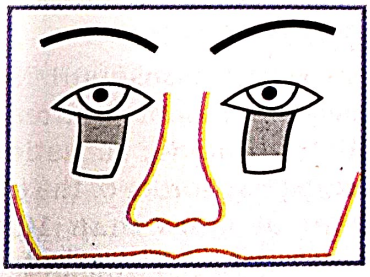
Paralysis of the Stapedius muscle causes an undue sensitivity to loud sounds which would normally elicit the protective mechanism. There is an apparent increase in loudness at high intensities of sound in the affected ear as compared to the normal ear.

ii. ***Stapedial Reflex***

Its absence is seen in Stapedius nerve involvement. Here loud sounds fail to elicit a change in the acoustic impedance, which is a very sensitive index.

4. ***Tests for Greater Superficial Petrosal Nerve***

Schirmer's Test – A strip of filter paper 5 cm length and 5 mm breadth is bent over at one end and hooked over the lower eyelid, on either side. Any impairment of lacrimation is evident if the wetting of the test strip on the affected side is markedly less than that on the normal side. A definite reduction of lacrimation suggests that the lesion involves the geniculate ganglion. This test is not relied upon, due to the following reasons that cause increased lacrimation on the affected side, inspite of facial paralysis.



Schirmer's test

Causes of increased lacrimation

i. Exposure Keratitis due to orbicularis oculi paralysis.

ii. Failure of drainage of tears due to eversion of lacrimal cannaliculi due to the loss of muscle tone.

iii. Crocodile tears, as a result of abnormal communication between the lesser and Greater superficial petrosal nerve.

Sequelae of Facial Paralysis:

1. ***Tics*** – Initially they are voluntary movements which mask a paralysis, later resulting into involuntary movements.
2. ***Mass movements*** – It is a loss of finesse of movements. This occurs due to absence of nerve shealth regeneration resulting in spread of nerve impulse to the other nerve fibres.
3. ***Synkinesis*** – Misdirection of the regenerating nerve fibres causes an unwanted group of muscles to contract.
4. ***Crocodile Tears*** – Misdirected nerve fibres resulting in an abnormal communication between the Greater and Lesser superficial petrosal nerve (VII & IX). This occurs after facial nerve paralysis due to Bell's palsy, herpes zoster oticus, fracture of temporal bone or intracranial surgical trauma. Therefore on eating, the parotid gland receives impulses which are then misdirected towards the facial nerve and this leads to tear forming via the greater superficial petrosal nerve to the Lacrimal gland. Treated by surgical resection of the tympanic branch of Glossopharyngeal nerve and by sectioning of the Vidian nerve.

Treatment

The aim is to prevent degeneration of nerve fibres.

I. *Medical*

1. *Oral prednisolone* – is the treatment of choice. 10mg tablet is given 4 times daily for 5 days and then gradually tapered within 3 weeks.

2. Vasodilator therapy - nicotinic acid is given orally.
3. Vitamins
4. Eye care - by use of spectacles (goggles) and eye drops acting as artificial tears.

II. *Physiotherapy*

Recommended to prevent contractures of the facial muscles.

III. *Surgical*

Facial Nerve decompression:

Indications

1. Acute Otitis Media with Facial palsy not responding to a Myringotomy.
2. Bells (Idiopathic Facial) Palsy showing degeneration of the nerve to be more than 95%.
3. Post-traumatic sudden, complete facial paralysis.
4. Iatrogenic Facial paralysis e.g. Post-Mastoidectomy, Stapedectomy etc.

Technique

1. Preferred to be done under General Anaesthesia as it may turn out to be a very lengthy procedure.
2. A postaural (Wilde's) incision is taken and a Simple Mastoidectomy performed.
3. The landmarks used to delineate the facial nerve are:
   i. Lateral Semicircular canal
   ii. Short process of Incus
   iii. Diagastric ridge and
   iv. Tympanic annulus.
4. Following the above landmarks the Facial nerve is exposed by clearing the
   i. Periosteal layer.
   ii. Plane of arteries and veins lying in loose connective tissue.
   iii. A fibrous layer, lying in contact with the perineural tissues.
5. On reaching the nerve, one of the following methods are performed.
   i. Draining an intraneural hematoma.
   ii. Removal of bony spicules pressing on the nerve.
   iii. Ends of the nerve are bought in close approximation.

Complications

1. Anaesthesia.
2. Conductive hearing loss due to ossicular chain disruption.
3. Labyrinthine damage.
4. Perichondritis.
5. Perforation of the Tympanic membrane.

Hemifacial spasms

These are unilateral, intermittent, involuntary, clonic contractions of facial muscles. The contractions increases after fatigue, stress and emotion.These begin in the orbicularis oris muscle and spread to the buccal muscles and platysma. Middle aged females are affected commonly.

Clinical features

Mild, progressive muscular weakness.

Intermittent spastic contractions of facial muscles which can be relieved by lying on the contralateral side.

Differential diagnosis

- Blepharospasm
- Habitual spasms.
- Primary Choleasteatoma
- Facial nerve neuroma.
- Acoustic neuroma.
- Neoplasms of ear and intracranium.
- Vertebral and basilar artery aneurysms

Investigations

- X-ray skull.
- Computerised tomography (Air contrast)
- Myelograms
- Electroencephalography
- Brain stem Evoked Response Audiometry

Treatment

Medical

- Dilantin
- Carbamazepine daily
- Phenobarbitone
  Dexamethasone
  Diamethylaminoethanol
  Diazepam

Surgical

- Micro-vascular decompression.
- Needling of the Facial nerve.

CHAPTER 20

Eustachian tube Diseases

Tubal Catarrh

Acute tubal catarrh occurs due to blockage of Eustachian tube by the untreated rhinitis, hypertrophied adenoids, sinusitis, nasal polyps, deviated septum or otitis media. It may occur following congested and edematous eustachian tube which gets narrowed and later gets blocked.

Clincal features

Symptoms

1) Ear blockage .
2) Pain
3) Deafness - conductive hearing loss
4) Tinnitus
5) Vertigo

Signs - Tympanic membrane is retracted and congested.

Treatment -

1) Antihistamines to the control rhinitis .
2) Decongestants
3) Valsalva's manouvre (not in the acute phase)
4) Surgical - Adenoidectomy, Septoplasty, Sinuscopy etc.

PATULOUS EUSTACHIAN TUBE

Here the eustachian tube is abnormally patent (too open a tube)

Causes

1) Idiopathic.
2) Sudden weight loss.
3) Pregnancy (last trimester)
4) Neurological disease (Multiple sclerosis)
5) Oral contraceptives
6) Exercise
7) Fatigue.

Clinical features

Symptoms -

1) Hearing of their own voice (Autophony)
2) Hearing once own breathing (due to transmission of respiratory noises to the ear)
3) Stuffiness in the ear
4) Tinnitus
5) Abnormality in speech

Signs – Otoscopy reveals movements of tympanic membrane with breathing.

It is due to loss of Ostmann's lateral pad of fat (present in the membranous part of cartilagenous Eustachian tube) or loss of fluid in the peritubal part.

Treatment

1) Reassurance. Self limiting.
2) In case of acute weight loss in persons, advise weight gain.
3) Intra-nasal or Systemic decongestants, Corticosteroids, Antihistaminics.
4) Cauterization of Eustachian tube (Chemical).
5) Systemic administration of iodides.
6) Grommet insertion.

SECTION - II

CHAPTER 21

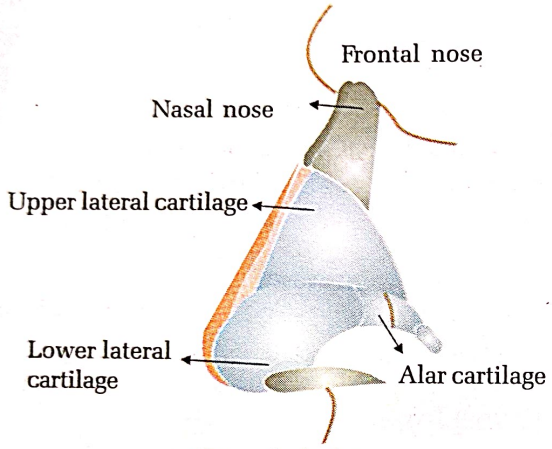
Anatomy of the Nose and Paranasal Sinuses

Anatomy of the Nose :

Shape – is pyramidal and maintained by the cartilagenous and bone framework.

The cartilagenous framework is formed by

i. Septal cartilage
ii. Two upper nasal cartilages
iii. Two lower nasal cartilages
iv. Two alar cartilages



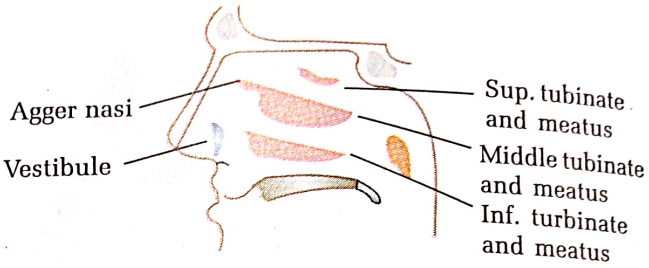
Nasal skeleton

Bony framework is formed by

i. Nasal bones
ii. Ascending process of the maxilla
iii. Nasal process of frontal bones

Nasal cavity – Of the right and left side is separated by the nasal septum. Each fossa communicates with the

i. Exterior - through anterior choanae
ii. Nasopharynx - through posterior choanae
iii. Paranasal sinuses - through their ostia

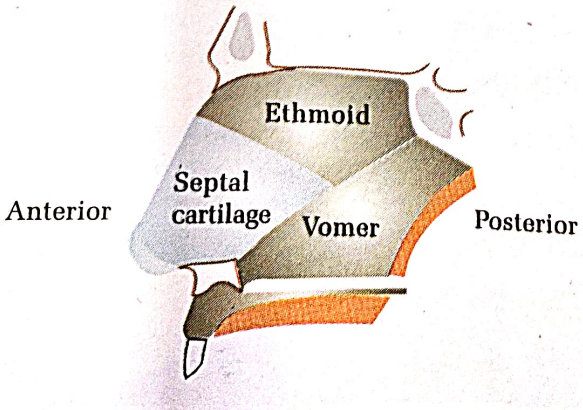


Structures on lateral wall of nose

I. ***Floor*** – is formed by
   1. Palatine process of the maxilla
   2. Horizontal parts of the palatine bone.

II. ***Roof*** – formed by
   1. Nasal process of frontal bone
   2. Cribrifrom plate of ethmoid
   3. Body of sphenoid

III. ***Medial wall*** – Which is the nasal septum. A concise knowledge of the nasal septum is necessary, before understanding septal surgery.



Medial wall of nasal cavity

Anatomy

1. ***Bony*** –
   i. Perpendicular plate of ethmoid
   ii. Vomer
   iii. Nasal Spine of Frontal bone
   iv. Rostrum of Sphenoid
   v. Crest of Nasal bones
   vi. Palatine bone

2. ***Cartilagenous***
   i. Septal Cartilage
   ii. Medial crus of greater alar cartilage

3. ***Membranous***
   Fibrofatty tissue at the anterior end of the septum

The three main constituents are
i. Septal cartilage
ii. Vomer
iii. Perpendicular plate of ethmoid

The nasal septum has four borders – Superior, Inferior, Anterior and Posterior and Two surfaces – Right and Left

IV. ***Lateral wall*** – Is formed mainly by the
1. Medial wall of maxilla
2. Lateral part of ethmoid and lacrimal bone.

The lateral wall has 3 parts.

1. Vestibule : It is a small depressed antero-inferior part of lateral wall which is lined by skin and contains "Vibrissae" a short, stiff and curved hairs.
2) Atrium – It is a hollowed out area opposite to the middle turbinate and above the vestibule of nose. Below it lies a ridge, i.e. Agger nasi runs downwards and forwards from the middle concha. Agger nasi some times contain agger nasi cells.
3. Turbinates – are posterior part of lateral wall.

The three turbinates are attached to the lateral wall of the nasal cavity. The superior and middle turbinates are a part of the ethmoid bone, while inferior turbinate is a separate bone by itself.

The three meatuses are named after the turbinates. Each meatus lies below and lateral to the corresponding turbinate.

Superior meatus : Small meatus, where posterior ethmoidal air sinuses open.

Middle meatus : Shows following features:-

Ethmoidal bulla – Rounded elevation of the anterior ethmoidal cells. Middle ethmoidal cells / sinus opens above the bulla.

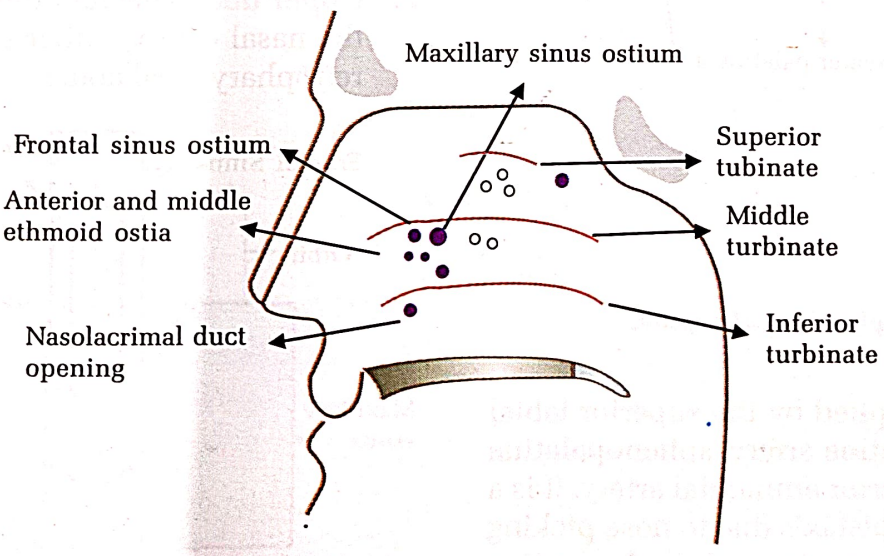
Hiatus semilunaris – Deep semicircular sulcus below the bulla and behind the uncinate process. In anterior part frontal sinus opens where as Maxillary sinus opens in the posterior part.

Ethmoidal infundilum – Short passage, in front of hiatus.

Uncinate Process – Thin bone parallel to the ethmoidal bulla which is attached inferiorly to the maxillary hiatus and superiorly may have an attachment to skull base or site of insertion of middle turbinate or lamina papyracea.

Inferior meatus : Mucosa covered Nasolacrimal duct opens in this large meatus. It can be identified by massaging the lacrimal sac at the medial canthus.

Sphenoethmoidal recess – This triangular fossa located superior to the superior turbinate. In this sphenoidal sinus opens.



Lateral wall of nose with tubinates removed and openings of sinuses

Relations of lateral wall

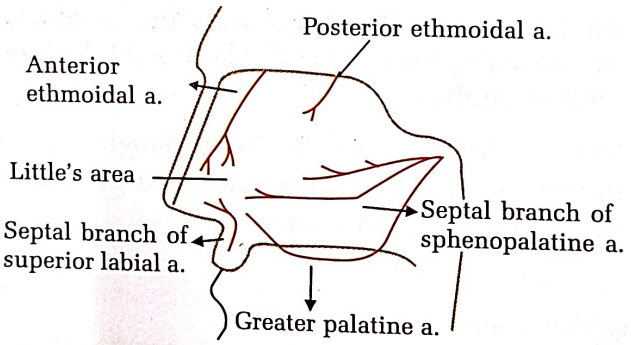
1. Above – separates ethmoids and orbit
2. Below – Maxillary sinus.
3. Front – Lacrimal groove and Nasolacrimal duct.

Blood Supply

The nose is supplied by branches of the

1. ***External carotid artery***
   a) Sphenopalatine a.
   b) Greater palatine a.
   c) Superior labial a.
2. ***Internal carotid artery***
   a) Anterior ethmoidal a.
   b) Posterior ethmoidal a.

They supply the nose, above the level of the middle turbinate.



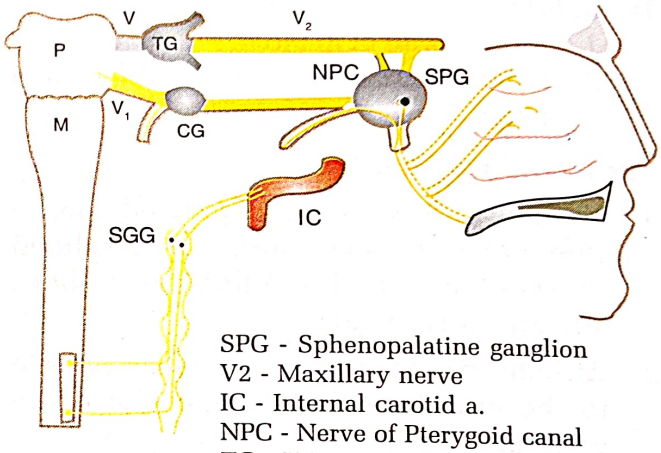
Blood supply of nasal septum

Little's area

Little's area is supplied by the superior labial artery, greater palatine artery, sphenopalatine artery and the anterior ethmoidal artery. It is a common site for epistaxis due to nose picking or trauma. The plexus of vessels is also known as Kiesselbach's plexus.

Nerve Supply

1. Trigemminal nerve (V) including the
   a. Anterior ethmoidal nerve
   b. Sphenopalatine ganglion and its branches
2. Olfactory nerve (I) - carry the fibres for smell
3. Autonomic nerve supply.

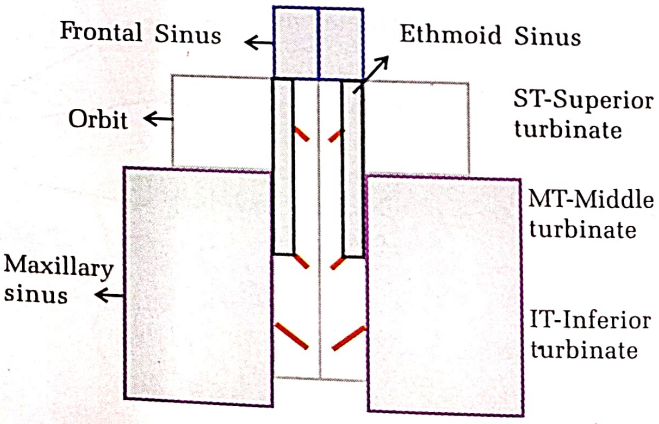


SPG - Sphenopalatine ganglion
V2 - Maxillary nerve
IC - Internal carotid a.
NPC - Nerve of Pterygoid canal
TG - Trigemminal ganglion
GG-Geniculate ganglion
SCG-Superior cervical ganglion

Autonomic nerve supply of nasal mucous membrane

Lymphatic drainage

1. ***Submandibular lymph nodes*** - drain the external nose and anterior part of the nasal cavity.
2. ***Upper deep cervical nodes*** - drain rest of the nasal cavity, either directly or via the retropharyngeal nodes.



Schematic representation of Paranasal Sinuses

PARANASAL SINUSES

These are air spaces in bones of the skull which are lined by mucous membrane continuous with that of the nasal cavity through their ostia.

1. ***Maxillary sinus*** – (Antrum of Highmore) is pyramidal in shape and occupies body of the maxilla. The base lies medially and apex in the zygomatic portion of the maxilla. It is the largest sinus, with an average capacity of 15cc. The roof of the sinus forms floor of the orbit and floor of the sinus is formed by alveolar process and the hard palate.

   Ostia – Opens into the middle meatus from the medial wall of the sinus.

2. ***Ethmoid sinus*** – Consists of a number of thin walled cavities (7-15) and are grouped as the anterior, middle and posterior ethmoidal sinuses.

   Ostia – Of the posterior ethmoidal sinuses drain into the superior meatus, while the anterior and middle ethmoidal sinuses drain into middle meatus.

3. ***Frontal Sinus*** – Occupies a very variable extent of the frontal bone and its average capacity is 7cc in the adult. The right and the left sinuses are often asymmetrical.

   Frontonasal duct – Opens into the middle meatus via the anterior ethmoidal sinuses.

4. ***Sphenoid sinus*** – lies behind the upper part of the nasal fossa with an average capacity of 7 cc in the adult.

   Ostia – Communicates with the superior meatus indirectly via the spheno-ethmoidal recess.

EMBRYOLOGY

Development : Appear as diverticulae from the nasal cavity. The diverticulae gradually invade the bones after which they are named. Maxillary and sphenoid sinuses begin to develop before birthwhile the other sinuses develop after birth.

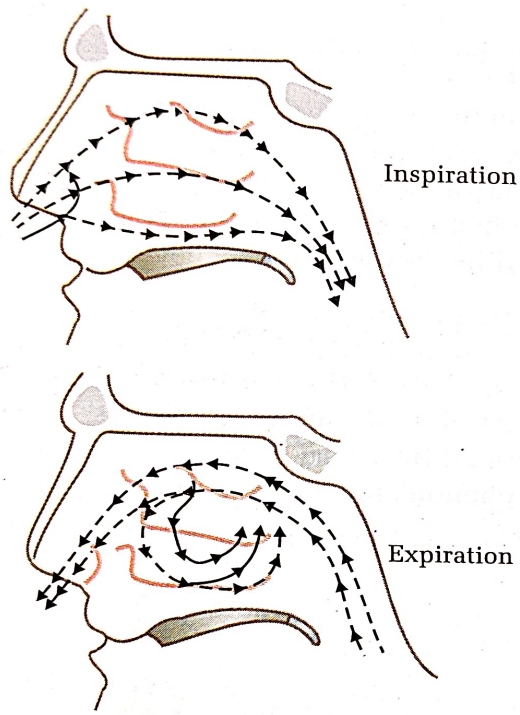
Growth of the sinuses continues during childhood and early adult life. Radiologically the Maxillary sinus can be identified at 4-5 months but is fully formed by 7 years , Ethmoids are formed by 1 year, Frontal sinus at 6 years and Sphenoid sinus at 4 years of age.

CHAPTER 22

Physiology of the Nose and Paranasal Sinuses

Functions of the nose :

1. **Respiration** – Is helped by the nasal passages as they constitute the uppermost part of the conducting passages along which, ventilation of pulmonary alveolar surface is maintained.



Physiology of nasal airflow

2. ***Air conditioning*** – Is important, as dehydration will deplete the mucous blanket which protects the cilia.
3. ***Filtration*** – Of noxious particles, micro-organisms etc. from the inspired air by vibrissae which filters coarse particles, cilia removes smaller particles by muco-ciliary clearance, lysozymes act as bactericidal agents and the mechanism of sneezing.
4. ***Olfaction*** – Has protective function, as well as causes reflex secretion of gastric juices. The olfactory sensitivity varies in individuals and depend on factors like olfactory threshold or the strength of odour.
5. ***Vocal resonance*** – Is necessary for normal speech and defect would lead to rhinolalia.
6. ***Speech*** – Production requires the nasal cavity as a resonator for a number of sounds. (The nose adds quality to the voice by allowing some air to escape through it. The sound resonates within the nose and mouth. In Rhinolalia clausa air escape will be minimum where as in Rhinolalia aperta air escape is maximum).
7. ***Nasal reflexes*** – Like sneezing, have a protective function.
8. ***Drainage*** – Of various sinuses and the nasolacrimal duct takes place in the nasal cavity.
9. ***Eustachian tube*** – Pressure regulation occurs through the nasal cavity.

Function of the Paranasal Sinuses :

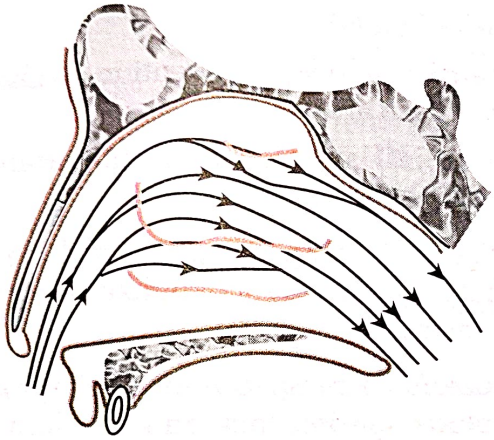
The following 'function' have been ascribed to these cavities.

1. ***Air conditioning*** – Of the inspired air by heating and moistening.
2. ***Vocal resonance*** – Imparts resonance to sound and this affects the quality of voice.
3. ***Thermal resonance*** – Is imparted to sound and this affects the quality of voice.
4. ***Vestigeal olfactory organs*** – As is in lower animals.
5. ***Formation is secondary to bony readjustments*** – e.g. Frontal sinus is formed by the disproportionate growth of face and cranium.
6. ***Aid to balance the head*** – By reducing weight of the bones of face.
7. ***Drainage*** – The nasolacrimal duct and paranasal sinuses drain into the nasal cavity.

Nasal Cycle

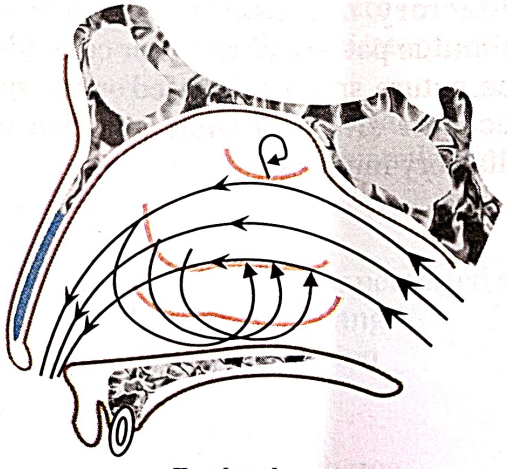
Nasal cycle is the alternate nasal blockage between the both sides. The physiological description was first given by Kayser. This cycle can be demonstrated by Rhinomannometry or by Thermography.

The direction of air flow during inspiration from nasal valve is upwards and backwards, above the inferior turbinate then it passes above and below the middle turbinate which rejoins in the posterior choanae.



Inspiration

On expiration the airflow becomes turbulent due to changing direction of airflow and calibre of the airway varies. The epithelial micro-anatomy and turbinates increases the surface area.



Expiration

The nasal resistance is made up by mucosa and muscles attached to bone and cartilages. Being a narrowest part the anterior valve promotes turbulent airflow. When the airflow is turbulent, the resistance is inversely proportional to the square of the flow rate. The nasal resistance transmits the positive pressure to the alveoli and keeps the lungs expanded during expiration.

(In tracheostomy the resistance is removed, which reduces the dead space and causes degree of alveolar collapse. This reduces alveolar ventilation and leads to right to left shunting of the pulmonary blood).

The nasal mucosa affects the airflow and nasal resistance.The mucosal changes are produced by the pseudoerectile tissue of veins of the nose. These changes takes place every 4 to 12 hours. The cycle is controlled by the autonomic nervous system. The nasal congestion is caused by the vagal overactivity. The nasal resistance is also reduced by inspiration of high levels of CO_2 by rebreathing, which is reversible by hyperventilation.

Factors modifying nasal cycle

Allergy, Infection, Exercise, Hormones, Pregnancy, Fear, Emotions like sexual activity or anger.

The flow of nasal secretion is also cyclical and secretions will be increased on the side of greater airflow.

Nasal secretions

The nasal secretions are produced by the mucous and serous glands. The mucous glands produce glycoproteins whereas serous glands produce water and ions. The glycoproteins are responsible for the viscosity and elasticity of mucus. The other secretions are enzymes (lysozymes, lactoferin), circulatory proteins (complement, A2 microglobulin. C-reactive protein), immunoglobulins (IgA, IgE, IgG ,IgM, IgD), cells (surface epithelium, basophils, eosinophils, leucocytes).

These keep the nose wet and have an antibacterial activity and are part of immune system.

OLFACTION

Physiology of olfaction

The olfactory cells are the smell receptors, present in the nasal mucous membrane. The olfactory cells are bipolar cells. The olfactory nerves are formed by the collection of unmyelinated axons.

The olfactory epithelium is situated in the cribriform plate, upper part of septum and superior part of turbinate. The immotile long cilia projecting from the bipolar olfactory neurons are concerned with odour reception. The primary olfactory neurons joins with mitral and tufted cells in the olfactory bulb, after passing through the cribriform plate. The intracerebral pathway involves the lateral olfactory tract, anterior olfactory nucleus, olfactory tubercle, the nucleus of the lateral olfactory tract, and the corticomedial nucleus of the amygdala. The cortical representation of olfactory processing in human beings is bilateral and situated in the pyriform cortex.

The olfactory epithelium absorbs the smell molecules and carries to the receptors on the cilia after binding with globulin and initiates membrane depolarization. The odour molecule binding causes allosteric changes in the receptors, this activates a stimulatory GTP - a binding protein which inturn activates the Adenyl cyclase and synthesize cyclic AMP by opening cation channel in the ciliary plasma membrane. This leads to depolarization and generation of action potential. The binding of odour molecule also causes activation of inositol triphosphate, which opens calcium channel in the membrane. This calcium binds to calmodulin and modultates the response of odour by amplifying the generation of cyclic AMP.

The olfactory epithelial cells are tuned for the identification of odour.

Every neuron responds to several odours. Various olfactory neurons combine to produce membrane depolarization which identify the odour.

The stimulation of a particular neuronal depolarization occurs for that particular odour which analyses and processes centrally, giving rise to a specific smell sensation related with that odour.

Theories of Smell

The theories of odour perception explain the nature. These are :

1. Moncrieff suggested the importance of molecular structure.
2. Briggs and Duncan postulated theory of photochemical reaction, like carotenoids, in the eye.
3. Mozell's Stereospatial theory the proposed a stereospatial, lock and key form. The firing of receptor cells occurs when the surface membrane changes.
4. Molecular properties – The basic molecular properties matter for receptor specificity and local polarization in the molecule. (Laffort, Patte and Etcmeto).
5. Olfactory mucosa morphology – The stimulus pattern of receptor cells identify the nature smell. It is based on the specific receptor site and their position in the olfactory mucosa.

CHAPTER 23

Examination of the Nose

History:

Nasal block - Duration
intermittent / continuous
unilateral / bilateral
diurnal variation
associated with headache
relief with nose drops

Nasal discharge - Duration
intermittent / continuous
unilateral / bilateral
watery / mucoid / purulent / blood stained
diurnal variation
associated with sneezing / headache

Nasal bleeding - Unilateral / bilateral
spontaneous / traumatic
number of episodes
quantity of blood loss
whether bleeding stops on its own bleeding from other sites treatment taken
family history

Headache - localized / generalised
dull / severe
with respiratory tract infection
diurnal variation
relief with analgesics

On Enquiry:

History of - Crusting
Absent or perverted smell
Nasal twang
Lacrimation
Fever
Earache
Ear discharge
Visual Disturbances
Cough
Trauma / nose picking
Allergy
Alcohol / snuff addiction

Nose Examination

I. ***External nose*** – Is inspected for
   i. Congenital deformity
   ii. Trauma
   iii. Scars of surgery
   iv. Broadening of nose due to a nasal mass
   v. Malignancy

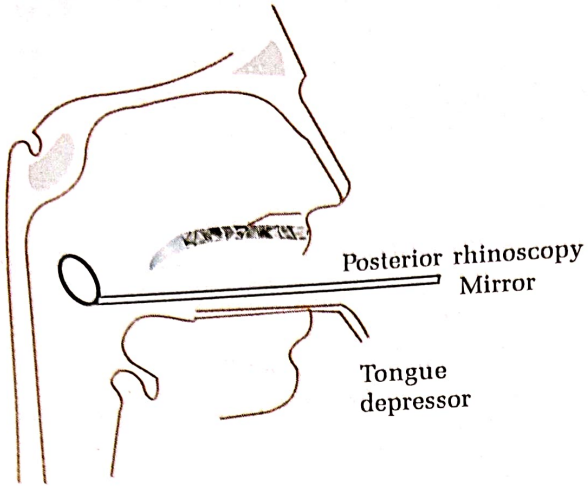
II. ***Anterior Rhinoscopy :***
   i. In children, the tip of the nose is lifted by the thumb of the examiner.
   ii. By using Thudicums nasal speculum, which is available in different sizes.

   The following structures seen are
   i. Nasal mucosa
   ii. Septum
   iii. Anterior end of inferior and middle turbinate.

The use of topical nasal decongestants allow clear examination. The nasal air blast can be checked by seeing for misting of a tongue depressor placed below the anterior nares.

III. ***Posterior rhinoscopy :***

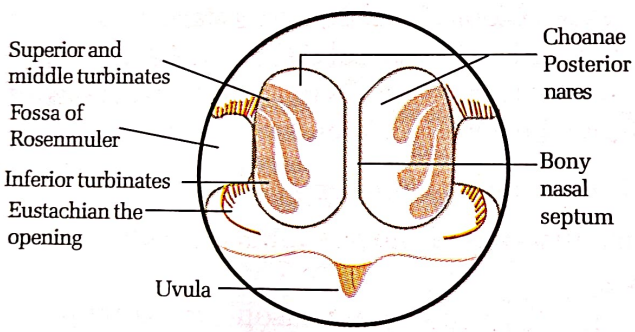
Visualisation of the nasopharynx can be done by a
   i. Yankauers rigid nasopharyngoscope
   ii. Fibre optic nasopharyngoscope
   iii. Posterior rhinoscopy mirror
   iv. Two lack's tongue depressors. The opposite end is used to elevate the soft palate and get a direct view of the nasopharynx.
   v. Two rubber catheters are introduced, each from either nostril and brought out of the mouth. On pulling the two ends together simultaneously, the soft palate gets everted to expose the nasopharynx.
   vi. Nasal Endoscopy.



Posterior rhinoscopy

Structures seen

i. Posterior choanae
ii. Posterior end of septum
iii. Eustachian tube orifices
iv. Roof of nasopharynx.
v. Posterior end of inferior and middle turbinates.



Structures seen on Posterior Rhinoscopy

IV. Paranasal sinus:

1. ***Inspection*** – for swelling, asymmetry and signs of inflammation to rule out tumours or osteomyelitis.
2. ***Palpation*** – for tenderness involving the
   i. Maxillary sinus – is tested above the canine fossa area.
   ii. Ethmoid sinus – is tested over sides of the nose, lateral to the inner canthus of the eye.
   iii. Frontal sinus – is tested just above the inner canthus of the eye.

Differential diagnosis of a Nasal mass

1. Antrochoanal Polyp
2. Ethmoidal Polyp
3. Hypertrophied inferior turbinate
4. Deviated (crooked) septum
5. Malignant growth
6. Rhinosporidiosis
7. Rhinoscleroma
8. Angiofibroma
9. Inverted papilloma
10. Meningocoele / Encephalocoele.

Antrochoanal Polyp	Ethmoidal Polyp
i. Single	Multiple
ii. Unilateral	Bilateral
iii. Infective	Allergic
iv. From the Maxillary Sinus	Ethmoid sinus
v. Children and young adults	Elderly
vi. Triradiate polyp	Single attachment
vii. Grow towards posterior choanae	Towards anterior choanae
viii. Recurrences rare	Very common
ix. Operation – Caldwell Luc.	Polypectomy and Ethmoidectomy

(3) Hypertrophied inferior turbinate :
Seen in adults
Deviated septum on the opposite side
Smooth surface
Arises from lateral wall of nose
Fixed and sensitive on touch
Shrinks with vasoconstrictors

(4) Deviated Septum :
Firm no movement
Sensitive to touch
Probe cannot be passed around

(5) Malignant growth :
Elderly patient
Reddish with slough , irregular surface
Hard and indurated, bleeds on touch

(6) Rhinosporidiosis :
Any age
Reddish with white spots
Mobile and soft
Bleeds on touch
No lymphnode involvement
Recurrence rate very high

(7) Rhinoscleroma :
Any age
Reddish in colour with nodular surface
Woody hard nose
Associated with palatal lesion
Fixed and firm

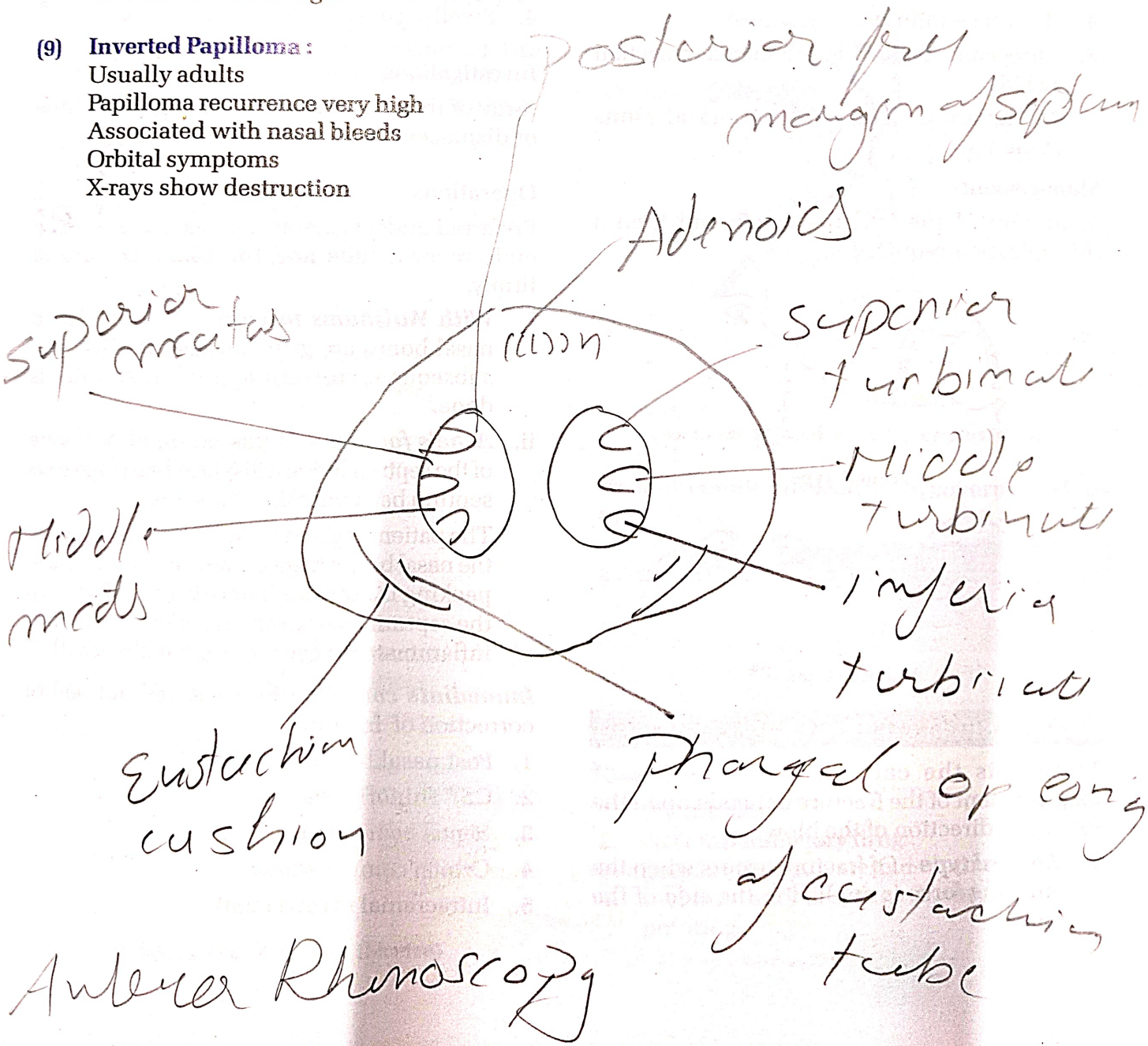
(8) Angiofibroma :
Usually young male
Smooth surface
Red colour
Bleeds on touch
No lymph node involvement
Arise from roof of nasopharynx
Recurrence rate is high

(9) Inverted Papilloma :
Usually adults
Papilloma recurrence very high
Associated with nasal bleeds
Orbital symptoms
X-rays show destruction

(10) Meningocoele/Encephalocoele
Younger age group
Neurological involvement
Transmitted cough impulse
X-rays shows deficient bone

Investigations:

1. X-ray Water's and Caldwell's view
2. Antral puncture
3. Nasal sinus endoscopy
4. Examination under general anaesthesia
5. CT Scan
6. MRI.



CHAPTER 24

Diseases of the External Nose

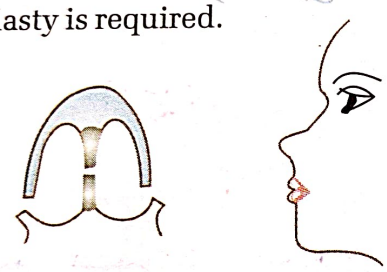
DEFORMITIES

Etiology

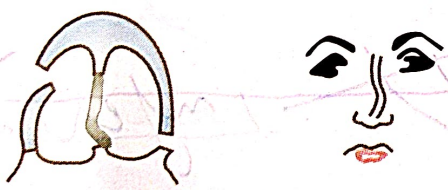
1. Congenital – Bifid nose or cleft lip.
2. Developmental – Along with deviated nasal septum.
3. Inflammatory – Septal abscess, leprosy or tuberculosis.
4. Trauma – injuries.
5. Iatrogenic – Following submucous resection (SMR).
6. Malignancy – Nasal or paranasal sinus tumours.

Management

Treatment of the existing cause and then a rhinoplasty is required.



Frontal type



Lateral type

FRACTURE NASAL BONES

Trauma is the cause and the degree of displacement of the fracture depends upon the force and direction of the blow.

I. ***Lateral type*** – Of fracture occurs when the injuring force is applied to the side of the nose.

II. ***Frontal type*** – Of nasal injury is more severe due to the injuring force being applied centrally from front.

Clinical features

1. Swelling , deformity and tenderness of the nose.
2. Nasal block and anosmia.
3. Epistaxis
4. Swelling over the face and eyelids.

Investigations

X-ray of the nasal bones, reveal a fracture line or displacement of the bones.

Operations

Preferred under General anaesthesia as with an endotracheal tube and the pharynx packed firmly.

i. ***With Walshams forceps*** – The displaced nasal bones are gripped, disimpacted and subsequent correction of the deformity is done.

ii. ***Ashe's forceps*** – Is passed on either side of the septum lifting a displaced cartilagenous septum back onto the vomerine crest.

The patient is given a plaster cast for keeping the nasal bones in place and an anterior nasal packing is done for hemostasis and to keep the septum in midline. Antibiotics and anti-inflammatory agents are given alongwith.

Immediate complications – Ruled out before correction of fracture

1. Post nasal bleed
2. CSF rhinorrhoea
3. Septal hematoma
4. Orbital complications
5. Intracranial involvement.

Late complications – of an untreated fracture of nasal bones.

1. Sinusitis
2. Thickened nasal bones
3. Atrophic rhintis
4. Synechiae.

VESTIBULITIS

It is a condition in which, skin of the nasal vestibule becomes excoriated and infected.

Etiology

1. Staphylococcus aureus is the causative organism.
2. Nose picking, excessively rubbing or cleaning the nose by the patient.
3. Dislocated columellar portion of the cartilagenous septum into the vestibule.
4. Diabetes may cause repeated furunculosis.

Clinical features

1. Pain
2. Swelling and cellulitis
3. Fever
4. Headache and malaise

Complications

1. Facial cellulitis
2. Cavernous sinus thrombosis
3. Blurring of vision or blindness.

Investigations

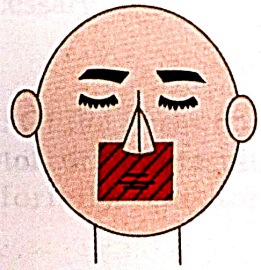
1. Blood sugar –diabetes.
2. Fundoscopy – papilloedema.

Management

No handling of the nose.

1. Antibiotics
2. Anti-inflammatory / analgesics
3. Local magnesium sulphate packs.
4. Local application of antibioticcs.

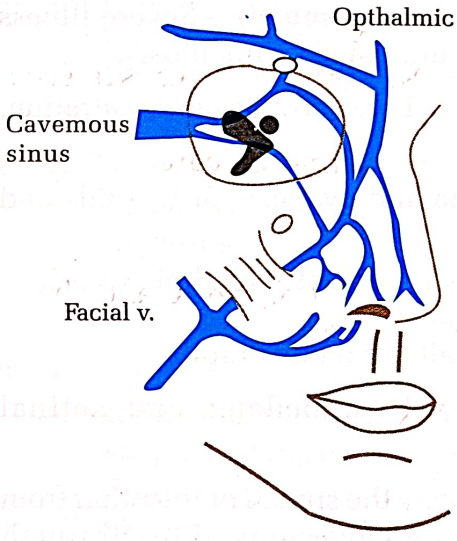
Danger area of the Face



Danger area of the face (Shaded)

Nasal furuncles are potentially dangerous because veins of the nose on each side drain into the facial veins, which like the opthalmic veins, have no valves. The opthalmic veins pass via the superior orbital fissure to the cavernous sinus. Squeezing or incision of the nasal furuncles are avoided unless definately pointing.

The first sign of Cavernous sinus thrombophlebitis is malaise, headache and fever. Tenderness along facial veins with lid edema occurs due to obstruction of the venous return from the opthalmic veins. Later, there is proptosis with restricted eye movement.



Veins associated with Cavernous sinus

Various conditions leading to cavernous sinus thrombosis

Source	Conditions
1. Nose/danger area	Furuncle/abscess
2. Pharynx	Tonsillitis/quinsy
3. Orbit	Cellulitis/abscess
4. Ethmoid sinus	Acute ethmoiditis
5. Sphenoid sinus	Sinusitis
6. Frontal sinus	Pyocoele/osteomyelitis

Treatment

1. Antibiotics
2. Anti-inflammatory drugs
3. Local antibiotic – steroid ointment
4. Incision and drainage if the abscess is pointing.

Furuncle – Acute infection of hair follicle.

These boils are tender and small. First appear at vestibule and may also involve tip, Fluctation may be present. Many furuncular nodules resolves after discharging into the vestibule.

Treatment

1. Intravenous antibiotics.
2. Local application of moist heat and antibiotic ointment.
3. Analgesics to relieve pain.
4. One should avoid squeezing the furuncle.

Complications

Cavernous sinus thrombisis – Severe illness caused by the untreated furunculosis.

Presentation – Fever, Vomiting, Prostration, Convulsions.

Signs – Edema and cyanosis of Eye lids and nose base.

Chemosis (obstruction of Ophalmic veins)
Opthalmoplegia
Dilated or small immobile pupils.

Late events – Papilloedema and Retinal haemorrhages.

This all occurs by the spread of infection from vestibule to the cavernous sinus through valveless facial veins and superior ophthalmic veins.

RHINOPHYMA

Rhinophyma is an enlargement of the nasal tip and nostrils, also referred to as a potato nose.

Pathology

It is caused by fibrosis and hyperplasia of the sebaceous tissues of the skin on the nose as a result of acne rosacea.

Treatment

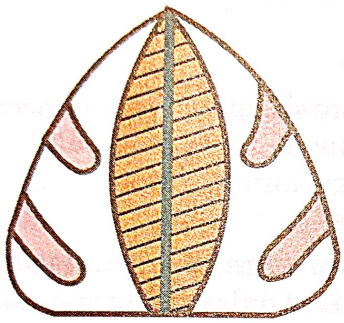
1. Shaving of excess tissue following which skin grafting required.
2. LASER therapy.
3. Cryosurgery.

CHAPTER 25

The Nasal Septum

SEPTAL HEMATOMA

When the septum is subjected to stress, submucosal blood vessels are torn and if the mucosa remains intact, this will result in the formation of a hematoma.



Septal Hematoma

Etiology

Following injury to the nose or following septal surgery.

Pathology

Blood accumulates in the subperichondrial layer and this will interfere with the vitality of the cartilage which is avascular and depends upon the perichondrium for its nutrition.

Treatment

An incision is made on the most dependent part of the hematoma. The cartilage if intact, bilateral incisions are necessary.

Complications:

1. ***External deformity :*** Of the noses due to absorption of the septal cartilage resulting into a supratip deformity and dorsal saddling of the nose.
2. ***Septal abscess***

SEPTAL PERFORATION

There is a communication between the two sides of the nasal cavities due to a breach in the nasal septum.

Causes:

1. ***Trauma***
   - i. Surgical – post SMR
   - ii. Repeated cautery
   - iii. Nose picking
2. ***Inflammatory***
   - i. Septal abscess
   - ii. Syphillis
   - iii. Tuberculosis
   - iv. Leprosy
3. ***Poison***
   - i. Industrial – chrome platers, painters, soap powders, etc.
   - ii. Snuff
   - iii. Cocaine
4. ***Malignancy***
   - i. Tumours
   - ii. Granuloma – Wegener's granuloma
5. ***Jewellery*** - is worn, for which septum is perforated.
6. ***Idiopathic***

Clinical features

1. Patient may be asymptomatic
2. Crusting at perforation site
3. Whistling sounds are produced with each breath in case of a small perforation
4. Dryness in the nose
5. Epistaxis.

Investigations

1. ***History*** – For diagnosing traumatic and occupational causes.
2. ***ESR*** – To rule out Wegeners granuloma and Tuberculosis.
3. ***Serological tests*** – For syphillis.
4. ***Biopsy*** – To execute malignancy.

Management

1. Treatment of the cause.
2. Alkaline nasal douches to clear the crusts.
3. Liquid paraffin nose drops to prevent crust formation.
4. Perforations upto 4 cm. in diameter are closed by various surgical techniques as mobilisation of local mucosal flaps.
5. Silastic obturators can be used to close any size of septal perforation. It is a simple, safe and reliable method.

DEVIATED NASAL SEPTUM

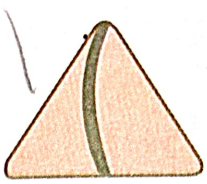
Etiology

Primary:

1. ***Direct trauma*** – Is the commonest cause of a septal deviation and may be associated with damage to other parts of the nose.
2. ***Birth moulding theory*** – Is applicable to patients with septal deviations where there is no obvious history of trauma . Dislocation and deviation of the septum was seen in primpara where second stage of labour lasted more than 15 min. Dislocation were to the right in case of LOA presentations and to the left with ROA presentations. Subsequent growth of the nose accentuates these asymmetries.
3. ***Hereditary.***

Secondary:

1. ***High arched palate*** – Results in buckling of the septum.
2. ***Mass*** – Tumour or polyp may push the septum to one side.



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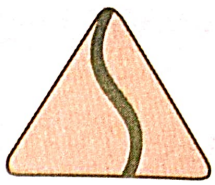

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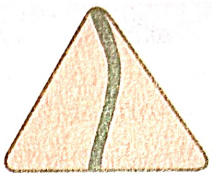
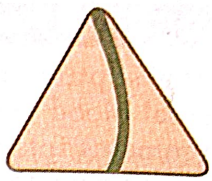


High deviation



Low deviation

Deformities:

1. **Spurs** are sharp angulations occuring at the junction of the vomer with the septal cartilage and ethmoid with the septal cartilage.
2. **Deviations** are characterised by a more generalised bulge. They may be 'C' shaped, 'S' shaped and usually involve both cartilage and bone.
3. **Dislocation** is when the lower border of the septal cartilage gets displaced from the central portion and projects into one of the nostrils.
4. **Thickened Septum** may be congenital or following trauma.

Symptoms

1. Asymptomatic
2. Nasal block
3. Vaccum headache
4. Recurrent colds
5. Anosmia
6. Epistaxis
7. External deformity.
8. Neuralgia.

Signs

1. External nose – may be deformed.
2. Anterior rhinoscopy – Reveals a deviation of the septum associated with turbinate hypertrophy on the opposite side.
3. Posterior rhinoscopy – Detects a posteriorly placed deviation.

Sequelae

1. Sinusitis
2. Otitis media
3. Atrophic rhinitis on the roomy side
4. Mouth breathing
5. Headache
6. Snoring
7. Asthma.

Differential diagnosis:

1. Septal hematoma
2. Nasal polyp
3. Hypertrophied turbinates.

Treatment

Septal surgery is required for symptomatic deviations. (SMR, septoplasty).

Indications for septal surgery

1. Septal deformities. All septal deformities do not require a correction. However, surgery is reserved for ones which interfere with nasal function.
2. Closure of septal perforations.
3. Graft material is obtained from the septum for operations like tympanoplasty.
4. As an approach to the pituitary –hypophysis.
5. As a part of Septorhinoplasty.
6. Easy access for polypectomy.
7. For easy access in endoscopic sinus surgery.
8. Before Palatoplasty.

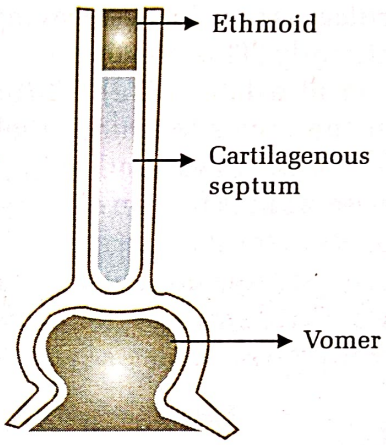
Contraindications

1. Age less than 16 years.
2. Acute rhinitis or sinusitis.
3. Hypertension.
4. Diabetes Mellitus.
5. Bleeding disorders.

Technique – SMR/Septoplasty

1. ***Anaesthsia*** – Preferred under local anaesthesia, as bleeding is minimal and results of air blast after correction of deformities can be checked on table. The nose is packed 10 min. prior to surgery with cotton pledgets dipped in 4% lignocaine with 1 in 2 lac dilution adrenaline. General anaesthesia is used in unco-operative patients or if rhinoplasty also is to be performed.

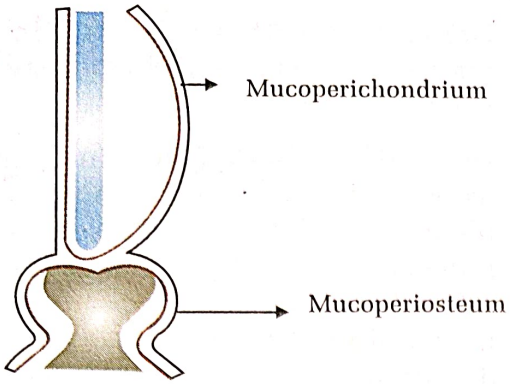
2. ***Infiltration*** – Of 2% lignocaine with 1 in 2 lac adrenaline in the submucoperichondrial plane, this is done till blanching of the mucosa occurs. The advantages of the infiltrations are :
   i. Local anaesthesia.
   ii. Bleeding is considerably reduced.
   iii. Tissue planes separation becomes very easy.



Nasal septum

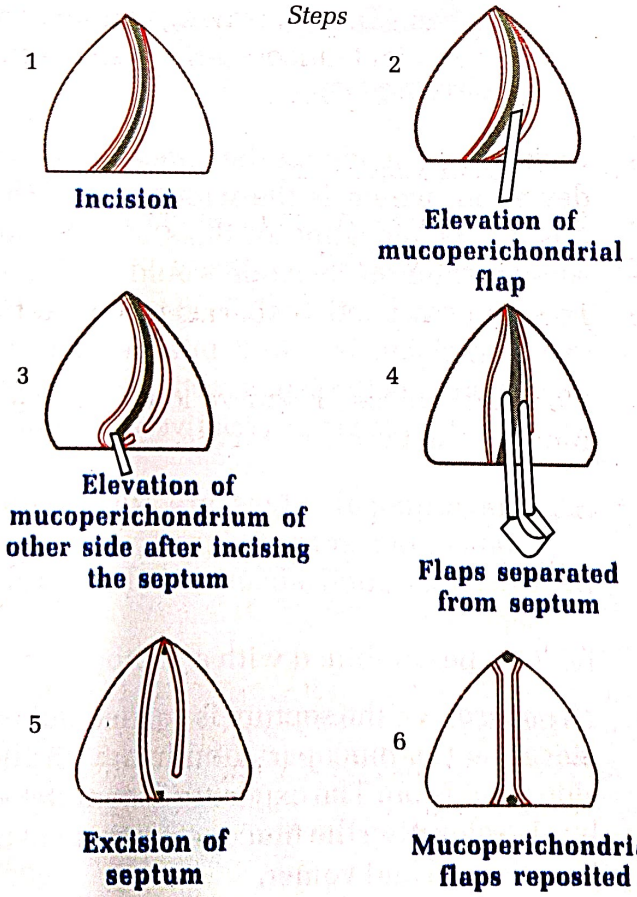
3. ***Incision*** – is made on the concave side of deviation, which is the wider side of the nostril. As, elevation of flaps beyond the site of maximum deviation would be a blind procedure and definately lead to a tear. The site of incision is 2 to 3 mm behind the lower border of the septal cartilage because.
   i. Here it is placed in a relatively avascular plane.
   ii. The mucosal edges are tough and chances of tear are reduced.
   iii. Provides good access to the whole septum.
   iv. Can be combined with a rhinoplasty.

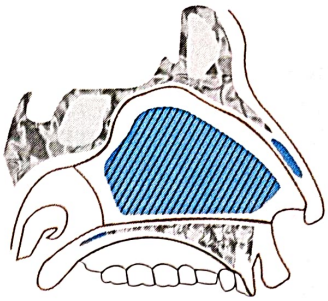
4. ***Exposure*** – Of the septum is carried out by elevating the mucoperichondrium on the side of incision. The exposure is completed by also elevating the mucoperiosteum over the ethmoid and vomer.



Elevation of mucoperichondrium and mucoperiosteum

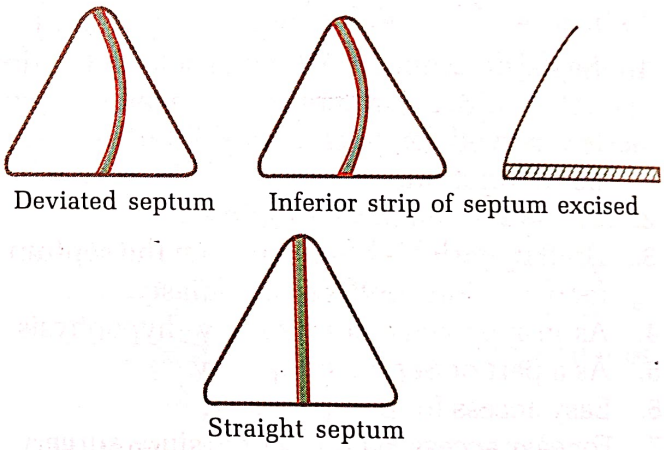
5. ***SMR (Sub Mucous Resection)***
The cartilage is now incised leaving a 5 mm strip anteriorly (This prevents a columellar retraction at a later date). Through this incision the opposite mucoperichondrial flap is elevated till the end of the septum. Now the septum is free on either side from the flaps. Leaving a 5 mm strip of cartilage superiorly (to prevent saddling of the nose) the septal cartilage is removed in a single action with the Ballenger's swivel knife.





Classical submucous resection of the nasal septum. The area resected is shaded

6. ***Septoplasty*** –Is a conservative SMR. Only the part of septum producing the defect is removed. Most of the septum is left intact, thus doing away with the problems of deformity of the nose occuring with the problems of deformity of the nose occuring with a SMR.



Septoplasty

The mucoperichondrial flap is elevated only on one (concave) side, the septal cartilage is freed from its bony attachment posteriorly. An inferior strip of cartilage is now removed, thus reducing the length of septum which can now be placed in the midline. Criss-cross incisions (not through and through) are taken on the cartilage, such that it loses its inherent elasticity and stays in midline.

6. ***Spurs,*** if any are removed with the help of
   i. Gouge and hammer.
   ii. Luc's forceps.
   iii. Bone punch.

7. Spatula test is performed. Misting by the air blast seen on the tongue depressor is an indication of adequacy of the air passages.
8. Two catgut stitches are taken at the incision site.
9. The throat is checked for postnasal bleeding.
10. With packs soaked in liquid paraffin, anterior nasal wall packing is done.
    The packs serve by :
    i. Keeping the Septum in the midline.
    ii. Preventing a Septum hematoma from forming.
    iii. Achieves hemostasis by pressure.

Post operative management

1. Antibiotics , analgesics and antihistaminics are given.
   Patient is given head–high position in bed to minimise the edema and vascular congestion.
2. The nasal pack is removed after 48 hours during which the patient breathes through his mouth.
3. Nasal washes are given to clear crusts formed due to dried blood clots.

Complications :

I. *Immediate*

i. Anaesthesia- cardiac arrhythmia , hypo or hypertension.
ii. Hemorrhage
iii. Tear of flaps
iv. CSF rhinorrhoea due to cribriform plate damage.

II. *Delayed*

i. Hemorrhage, this usually stops by gelfoam packing only.
ii. Septal hematoma.
iii. Septal abscess - This results from an infected septal hematoma. The abscess is drained immediately and patient is kept under injectable antibiotic cover. Any delay in drainage could lead to Cavernous sinus thrombosis.
iv. Septal perforation resulting from a tear on either side of the flap at the same site.
v. Meningitis, as this area falls in the region of danger area of the face. Infection travels intracranially through the veins which are valveless causing Frontal lobe abscess.
vi. Adhesions occur in the nasal cavity as a result of damage to surrounding structures causing raw area formation. The adhesions are broken and a splint is introduced to keep the raw surfaces away.

III. *Late*

i. *Atrophic rhinitis*, if extensive mucosal damage has occured.
ii. Columellar retraction, if no anterior strip of cartilage is left during an SMR.
iii. Supra-tip deformity and saddling of the nose, again if no cartilage strip is left superiorly. Not seen in septoplasty.
iv. Flappy septum occurs due to excessive cartilage removal, leaving behind a lot of redundant mucosa which falls to one side on lying down.

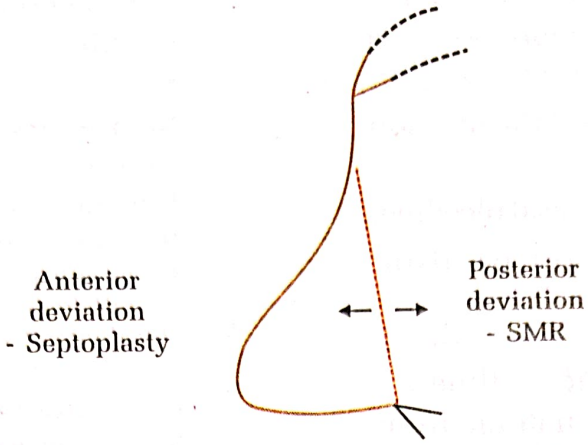
Advantages of Septoplasty

1. Can be done in the younger age group.
2. Done along with Rhinoplasty
3. Prevents any Post-operative cosmetic deformity.
4. No chances of Septal perforation, as one side mucoperichondrium remains intact.

Choice of Technique

Septoplasty is performed for an anterior deviation, while SMR for posteriorly placed deviation.

An imaginary line passing from the nasal process of maxilla to the nasal process of frontal bone is used to determine whether a septoplasty or SMR is required.



Septoplasty or SMR

	SMR	SEPTOPLASTY
1. Operation	Radical	Conservative
2. Age	Above 16 years only	Any age
3. Flaps	Elevated on both sides	Only on concave side
4. Caudal dislocation	Not corrected	Corrected
5. Cartilage removal	Except 2 mm strip Caudal end and roof	Only excess (minimal)
6. Complications	Septal haematoma CSF leak Septal perforation Flappy septum Columellar retraction Supra tip deformity Saddle nose	Rare
7. Rhinoplasty	Not done	Can be combined

CHAPTER 26

Infections of the Nasal Cavities

Infective Rhinitis

1. **Acute**
   a) Non-Specific-Common cold
   b) Specific – Diphtheria
2. **Chronic**
   a) Non-Specific – simple chronic rhinitis
   b) Atrophic rhinitis
   c) Hypertrophic rhinitis
   d) Rhinitis sicca
   e) Rhinitis caseosa
   f) Malignant granuloma

Specific

1) Rhinosporidiosis,
2) Rhinoscleroma
3) Syphilis
4) Leprosy
5) Tuberculosis,lupus vulgaris
6) Fungal infection like aspergillosis, actinomycosis,moniliasis
7) Leishmaniasis
8) Acute rhinitis can be viral, bacterial or irritative.

COMMON COLD (CORYZA)

It is the commonest viral infection in man. The incidence estimated, is that every adult has two to three colds a year. Children of early school age are most susceptible.

Predisposing factors

1. Chill and humidity
2. Lack of exercise
3. Dietary hypersensitivity
4. Nasal obstruction
5. Vitamin deficiency
6. Focus of infection
7. Generalised disorders e.g. diabetes.
8. Hypo or hyperthyroidism.

Causative organisms

It is generally accepted that common cold occurs due to infection with filterable viruses, followed by secondary infection by bacteria.

i. Influenza virus
ii. Rhino virus
iii. Respiratory synctial virus
iv. Adeno virus
v. Parainfluenza virus

Mode of transmission

i. Droplet and dust
ii. Droplet and nuclei
iii. Contact

Defences

i. Vibrissae in the nasal cavity
ii. Mucous blanket
iii. Epithelial cells
iv. pH of secretions
v. Interferon

Clinical features

1. ***Prodromal or ischemic stage*** – lasts for a few hours with feeling of tickling in the nasal cavity.
2. ***Early reaction and irritation*** – occurs as infection spreads to the adjacent mucous membrane and there is watery discharge, sneezing and nasal block.
3. ***Venous stasis and secondary infection*** – results into a purulent discharge and the mucous becoming dusky. Nasal obstruction and toxaemia is maximum.
4. ***Resolution*** – occurs with the symptoms and signs gradually diminishing and after 5-10 days recovery takes place.

Differential diagnosis -

1. Allergic rhinitis
2. Influenza rhinitis

Complications

1. Nasopharyngitis and pharyngitis
2. Sinusitis
3. Acute otitis media
4. Tonsillitis
5. Lymphadenitis
6. Bronchitis and asthma,pneumonia
7. Gastroenteritis

Management

1. Rest
2. Avoiding fatigue and sudden change of temperature
3. Vitamin C to improve resistance
4. Analgesics and anti-pyretics
5. Antibiotics in the presence of secondary infection
6. Nasal decongestants

NASAL DIPHTHERIA

It is an acute infection of the nasal cavity due to the organism Corynebacterium diphtheriae.

Clinical features

Nasal diphtheria may be primary or secondary to the faucial form. The symptoms being blood stained nasal discharge and fever.

Treatment

1. Anti-toxin to neutralise the toxin.
2. Antibiotics.
3. Isolation is continued till three successive daily swabs are negative.

ATROPHIC RHINITIS

It is a chronic nasal disease characterised by progressive atrophy of the mucosa and turbinates with the presence of viscid secretions which rapidly dry and form crusts emiting a foul odour called **ozaena** (*stench*).

Etiology

The exact cause remains unknown. The organisms cited as the cause are

1. Coccobacillus
2. Klebsiella ozaena.
3. Diphtheroid bacilli
4. Coccobacillus foetidis ozaena.
5. Bacillus mucosis.

The various other etiological factors being

i. ***Age*** – Commonest in the 2nd decade
ii. ***Sex*** – Seen predominantly amongst females
iii. ***Familial*** – Autosomal dominant trait
iv. ***Anatomical*** – People with wide and short noses are more affected
v. ***Endocrine*** – Peak incidence is found at puberty and disease is worse during menstruation.
vi. ***Infections*** – Chronic sinusitis, rhinitis etc.
vii. ***Trauma*** – Post SMR, injury etc.
viii. ***Vitamin A deficiency***
ix. ***Malnourishment***
x. ***Autonomic imbalance***.

Secondary atrophic rhinitis may occur following leprosy, syphilis or tuberculosis. However, rarely seen.

Pathology

1. **Mucosa** – Of the nose undergoes atrophy, which then appears pale with crusts and secretions.
2. **Turbinates** – Are shrunken, resulting into a roomy cavity.
3. **Blood vessels** – Show endarteritis and periarteritis.
4. **Nerves** – Become atrophied.

Symptoms

i. ***Nasal obstruction*** – Due to crusts which blocks the nose.
ii. ***Foul odour*** –Leads to social problems for the patient.
iii. ***Epistaxis*** – Following removal of crusts.
iv. ***Crusting*** – Due to drying of the nasal mucosa.
v. ***Drying*** – Following destruction of the turbinates.
vi. ***Anosmia*** – Because of atrophy of the nerves.

Examination:

1. ***Externally*** – There may dorsal saddling of the nose due to atrophy of the septal cartilage.
2. ***Anterior rhinoscopy*** – Reveals pale mucosa, greenish crusts, atrophic turbinates with a roomy nostril.
3. ***Posterior rhinoscopy*** – May show crusts.

Investigations

1. ***Hemogram*** – Anemia
2. ***Dermatological examination*** – Leprosy and syphilis
3. ***VDRL*** – Syphilis
4. ***X-ray paranasal sinuses*** – Sinusitis
5. ***X-ray chest*** – Tuberculosis
6. ***Antral puncture*** – Sinusitis
7. ***Nasal smear*** – For leprae or tubercle bacilli.
8. **Culture** of nasal secretions.

Differential diagnosis:

1. ***Sinusitis*** – Foul smell may be present but crusting is absent.
2. ***Chronic infections*** – Like leprosy, tuberculosis or syphilis.
3. ***Rhinolith*** – Is always unilateral.
4. ***Scleroma*** – Shows atrophic changes during the earlier stages of the disease however a biopsy would confirm the diagnosis.
5. ***Rhinitis sicca*** – Is characterised by crusting only in the anterior part and there is no foul odour.

Management

I. Local :

(1) Alkaline Nasal douche

Constituents

i. Sodium bicarbonate (28.4 g) – Creates an alkaline medium which is necessary to dissolve the crusts.
ii. Sodium biborate (28.4g) – Acts as an antiseptic.
iii. Sodium chloride (56.7 g) – Makes it an isotonic solution.

The patient is instructed to take nasal washes twice a day with one teaspoonful of the above powder in a pint of water.

(2) *25% Glucose in glycerine nose drops*

i. Glycerine (30 cc) – Prevents drying of the nasal mucosa.
ii. Glucose (8 g) – Inhibits the growth of proteolytic organisms.

II. Systemic :

1. Antibiotics to control infection.
2. Vitamin A supplements.

III. Surgical:

1. ***Young's operation*** – Involves closing the nostrils by raising mucosal flaps and suturing them. This gives rest to the mucosa and crusting is prevented as no air passes through.

   Modified Young's Operation – Involves partial closure of nostril leaving a 3mm hole. This relieves mouth breathing and nasal voice and can be well tolerated by most of people.

2. ***Implantation*** – Of fat, placental extracts, bone chips etc, so as to reduce the size of the nasal cavity. (Teflon strips, Polythene and Cartilage).
   - Local injection of liquid Paraffin into submucosa to displace lateral nasal wall medially.
3. ***Lautenslager's operation*** – Is medialisation of the lateral wall of the nose.
4. ***Wittmack's operation*** – is implantation of the Stenson's duct into the nasal cavity.
5. ***Raghav Sharan's operation*** – Is transplantation of antral mucosa into the nasal cavity.
6. ***Stellate ganglion blocks*** – To improve nasal blood supply.
7. ***Cervical sympathectomy or blockade***

Complications

The disease may persist for long periods but regresses on its own. However, the sequelae are

1. Chronic sinusitis
2. Atrophic pharyngitis
3. Otitis media
4. Myiases – maggots in the nose
5. Social stigma – on account of the stench or foul odour.

RHINOSPORIDIOSIS

It is a chronic infestation by yeast like fungus Rhinosporidium seeberi and Kineayli of the mucous membrane of the nose and nasopharynx.

Etiology

1. ***Age*** – Seen in young adults.
2. ***Sex*** – Mostly among males.
3. ***Geographical distribution*** – Predominantly occurs in south India.
4. ***Trauma*** – Is a predisposing cause, as occurs after an abrasion.
5. ***Occupation*** – Commoner in agriculture workers.
6. ***Miscellaneous*** – Pond bathing is common in these patients.

Pathology

The characteristic lesion is a bleeding polypus. Histologically the polypus has a vascular fibromyxomatous structure and throughout cells containing the sporangium are seen.

Clinical features

1. The polypoidal mass causes nasal block and headache.
2. Epistaxis occurs as the nasal mass is very friable.
3. Anterior rhinoscopy reveals a strawberry like mass with greyish surface studded with multiple sporangia seen as white dots.
4. Nasal infection may then lead to a secondary infection of the trachea, bronchus and the nasolacrimal sac.

Management

1. *Dapsone (100 mg)* twice daily. It increases fibrosis, reducing the bleeding and recurrence chances are minimised by preventing dissemination of the sporangia during surgery.
2. *Surgical excision* – is the treatment. The base of the polypus is cauterised by a diathermy knife to prevent bleeding and recurrence.

SCLEROMA

It is a chronic, progressive disease which begins in the nose and spreads to the pharynx, larynx and rarely to the tracheobronchial tree.

Etiology

1. ***Age*** – Seen in adults
2. ***Sex*** – No predilection
3. ***Occupation*** – Common in farmers
4. ***Organisms*** – Klebsiella rhinoscleromatis (Frisch's bacillus).

Pathology

Histologically granulomatous tissue infiltrates the submucosa and is characterised by plasma cells, lymphocytes, large foam cells (Mickulicz cells) which have a central nucleus and a vacuolated cytoplasm containing Frisch's bacillus and Russell bodies.

Clinical :

1. ***Atrophic stage*** – The changes in the mucosa resemble that of atrophic rhinitis.
2. ***Nodular stage*** – Development of pale and hard non-ulcerative nodules occurs.
3. ***Cicatrizing stage*** – Is characterised by stenosis and adhesions that distort the normal anatomy of the nasal cavity.

Diagnosis:

1. Painless condition.
2. External nose gives a 'woody' feel.
3. Various clinical stages are intermingled with each other.

4. Uvula sign – The uvula is retracted (fibrosis) or even absent.
5. Biopsy.

Differential diagnosis

1. Atrophic rhinitis
2. First stage of acute rhinitis
3. Keloid
4. Kochs
5. Malignancy
6. Leprosy.

Treatment

1. ***Antibiotics*** – Tetracycline , Streptomycin , Chloromycetin or Ampicillin is given. Useful only in the early stages of nasal lesions.
2. ***Steroids*** – Are used for preventing further fibrosis, as cannot reduce the fibrosis already present.
3. ***Local injection*** – Of carbolic acid and glacial acetic acid with glycerin mixture, to achieve regression of the granulomatous lesion without scarring – Kailash Rai regime.
4. ***Surgery*** – Performed when fibrosis has already occured. The fibrotic tissues are excised and nasal airway restored. Surgery is contraindicated during active disease.

Rhinitis sicca

It is a form of atrophic rhinitis. Here the nose is dry and shows mild atrophy which does not progress into atrophic rhinitis. The ciliated epithelium changes to cuboidal or columnar epithelium. The muco-ciliary dysfunction leads to loss of mucous blanket movement ability. The seromucinous glands become atrophied.

Etiology :

i) Alcohol consumption
ii) Anaemia
iii) Malnutrition
iv) Idiopathic

Common in people who work in dry, dusty and hot environment.

Clinical features

Symptoms

i) Epistaxis
ii) Discomfort
iii) Irritation

Signs

i) Mucous membrane is dry and white
ii) Septal perforation with thin, dry crusts.
iii) Bleeding on removing crusts.

Investigations : Same as Atrophic rhinitis.

Treatment

i) Treat the underlying cause.
ii) Supplementation of Iron and Vitamins.
iii) Local douching by Alkaline solutions.

Rhinitis Caseosa (Nasal Cholesteatoma)

This is a chronic inflammatory nasal condition. Here granulation tissue forms and Cholesteatoma like foul cheesy material collects in the nose. Commonly it involves one side of nose and males are affected.

Etiology

i) Idiopathic
ii) Syphilis
iii) Cholesteatoma
iv) Degeneration of polyps
v) Tuberculosis

According to the theory of suppurative rhinitis the underlying pathology (like DNS, rhinoliths, Polyps, Inflamed turbinates) complicated by obstruction of nasal secretions cause collection of discharge with superadded sinus infection.

Clinical features

Symptoms

i) Blocked nose
ii) Abscess on face
iii) Fistula

Signs

i) White debris
ii) Inflammation of face
iii) Signs of bone invasion

Investigations

1. ***Complete blood count*** – Anaemia .
2. ***X-ray of Paranasal sinuses*** – To know underlying sinus infection and destruction of bone.
3. ***Histology*** – To know the underlying condition.

Treatment – Surgical debridement of granulation tissue and daily vigorous cleaning of nose. Complete removal is important.

Treat the underlying infection with antibiotics.

CHAPTER 27

Allergic and Vasomotor Rhinitis

ALLERGIC

Allergic rhinitis is a condition giving rise to one or more symptoms of nasal obstruction, increased secretion of mucus and sneezing due to an altered reaction to an antigen (allergen).

Etiology

1. ***Age*** – The peak incidence is between 11-30 years.
2. ***Sex*** – Equally affecting both.
3. ***Famillal*** – Higher chances if both parents have allergy.
4. ***Climate*** – Precipitated by cold air, as it stimulates the nasal glands and reduces nasal patency.
5. ***Psychological factor*** – May only accentuate allergy as a precipitating factor.

Allergens

1. Inhalants – Dust, feather, pollen etc.
2. Food – Egg, milk, fish, citrus food etc.
3. Drug – Aspirin, hypotensive drugs etc.
4. Cosmetics and powders.
5. Irritant – Fumes and smoke.
6. Intestinal helminths.

Types :

1. ***Seasonal allergic rthinitis*** – Occurs due to pollens, flowers and shrubs.
2. ***Perrenial allergic rhinitis*** – Is due to house dust, milk, eggs etc.
3. ***Polyps*** – May be present.

Investigations

1. ***Detailed personal history*** – Allergy
2. ***Anterior rhinoscopy*** – Septal deviation, nasal polypi and hypertrophic mucous membrane
3. ***X-ray paranasal sinuses*** – Sinusitis and polyps
4. ***Nasal secretions*** – Eosinophilia
5. ***Stool examination*** – Helminths
6. ***Intradermal skin tests*** – Allergen detection
7. ***Nasal provocation tests*** – Inhalant allergies
8. ***RAST*** – Radioallergosorbent test to detect IgE antibodies to particular antigens.

Management

1. ***Avoidance*** – Of allergens
2. ***Desensitisation*** – By vaccines
3. ***Drugs***
   i. *Anti-histaminics* – Relieves symptoms by decreasing secretions.
   ii. *Corticosteroids* – Help by their anti-inflammatory, antiallergic action.
   iii. *Sodium chromoglycate* – Prevents degranulation of mast cells thus prevents release of histamine.
4. ***Local*** – At the inferior turbinate.
   i. Silver nitrate application
   ii. Submucous diathermy
   iii. Auto-haemo injections
   iv. Corticosteroid injections
   v. Cryosurgery
5. ***Surgical***
   i. Correction of septal deviation and removal of polypi to reduce the symptoms.
   ii. Vidian neurectomy.
   iii. Septic foci removal – e.g. Tonsillectomy or dental extraction.

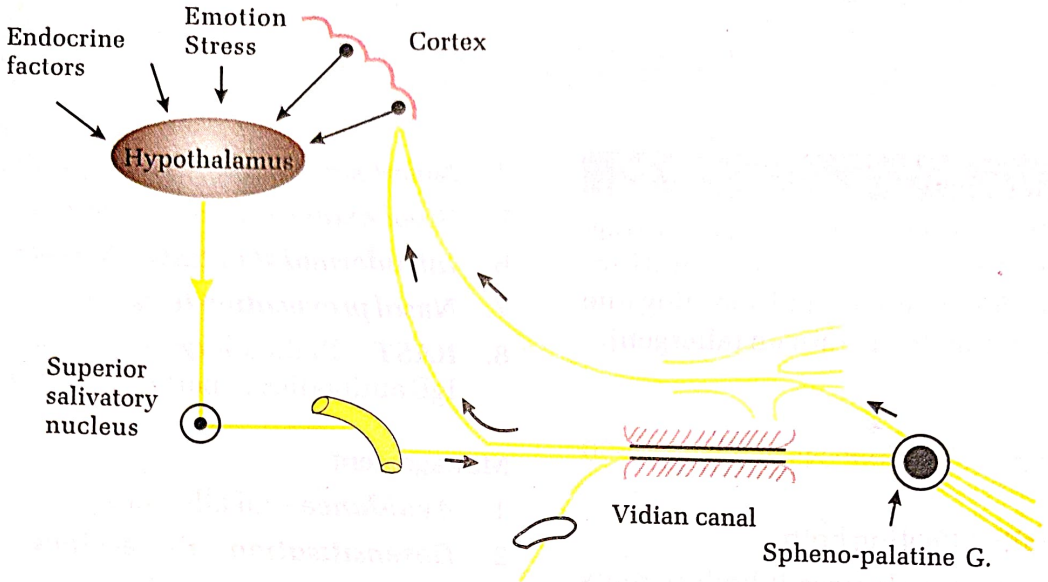
VASOMOTOR RHINITIS

Here, the nasal mucosa is hyper-reactive to certain stimuli in the absence of identifiable allergic causes.

Etiology:

Predisposing factors

1. ***Hereditary.***



Neural mechanisms of Secretomotor rhinopathy

2. ***Infection*** – A preceding history of infection (bacterial or viral is common).
3. ***Septal deviation*** – Make the obstructive symptoms worse.
4. ***Psychological and emotional factors*** – As anxiety,resentment, grief or tension may be responsible.Treatment by counselling or drugs like antidepressants or tranquillisers.
5. ***Endocrine influence*** - Seen particularly at puberty, during menstruation, pregnancy and as honeymoon rhinitis. Following the hormonal changes the nasal mucosa becomes edematous and blocks the airway.
6. ***Drugs*** - Hypotensive agents like reserpine, guanethedine or propranolol cause edema of the nasal mucosa.Contraceptive pills also lead to nasal block because of estrogens.
7. ***Overuse of local applicants*** – This tends to produce 'rhinitis medicamentosa' due to rebound mucosal congestion. Treated by withdrawl of drugs, steroid therapy or surgical reduction of hypertrophied turbinates.
8. ***Hypothyroidism*** - Causes a reduced activity of the sympathetic system resulting into nasal block and stuffiness.Replacement by thyroxine relieves the condition.

Precipitating factors

1. Atmospheric conditions like change in temperature and humidity.
2. Reflex sneezing during exercise or getting onto a cold floor.
3. Fumes, dust and alcohol.

Pathology

The nasal mucosa is hyperaemic, polypi may be seen and the inferior turbinate is hypertrophied.

Clinical features

It affects all ages.

1. Sneezing is paroxysmal.
2. Rhinorrhoea is profuse and watery.
3. Nasal obstruction, may alternate with positional changes from side to side.
4. Postnasal discharge.

Differential diagnosis

1. Allergy
2. Infection
3. Foreign body, adenoids in children.

Management

1. Avoidance of precipitating factors
2. Anti – Histaminics
3. Topical steroid spray
4. Chemical cauterisation of sensitive mucosal areas.
5. Submucosal diathermy
6. Cryosurgery
7. Polypi removal
8. Septal correction
9. Vidian neurectomy
10. Psychological adjustments, sedatives and tranquilizers may be needed.

	Allergic Rhinitis	Vasomotor Rhinitis
i. Sneezing and rhinorrhoea	Seasonal and perrenial	Frequent
ii. Nasal mucosa	Bluish	Swollen
iii. Lacrimation	Usual	At times
iv. Eosinophilla	Marked	Less intense
v. Skin tests	Positive	Negative
vi. Response to anti-histaminics	Good	Variable
vii. Polyps	Frequent	Occasional
viii. Associated asthma	Frequent (40%)	Occasional

CHAPTER 28
Epistaxis

Epistaxis is bleeding from the nose

CAUSES

I. *Local :*

1. **Congenital**
   - Osler Weber Rendu disease. (hereditary hemorrhagic telangiectasis)
2. **Traumatic**
   - **A) Breach in mucosal continuity**
     i. Fractures of nose
     ii. Operations e.g. SMR, polypectomy etc.
     iii. Nose picking.
   - **B) Barotrauma**
     i. Nose blowing
     ii. High altitudes
3. **Inflammatory**
   - **A) Acute**
     i. Non-specific rhinitis e.g. coryza.
     ii. Specific rhinitis e.g. diphtheria, adenoiditis, sinusitis etc.
   - **B) Chronic**
     i. Non- specific e.g. atrophic rhinitis
     ii. Specific e.g. scleroma, Rhinosporidiosis, chronic sinusitis etc.
     iii. Midline nasal granuloma
4. **Neoplastic**
   - **A) Benign**
     i. Angiomatous polyp
     ii. Papilloma
     iii. Nasopharyngeal angiofibroma
   - **B) Malignant**
     i. Carcinoma
     ii. Sarcoma of the sinuses/nasopharynx
5. **Miscellaneous**
   - i. Foreign body
     ii. Rhinolith
     iii. Nasal parasites

II. **Systemic :**

1. **Cardiovascular system**
   - i. Hypertension
     ii. Rheumatic Heart disease
     iii. Pulmonary Hypertension
     iv. Infective Endocarditis
2. **Blood disorders**
   - i. Christmas disease
     ii. Hemophilia
     iii. Purpura
     iv. Aplastic anemia
3. **Drugs**
   - i. Salicylates
     ii. Quinine
4. **Renal failure**
5. **Hepatic failure**
6. **Vicarious menstruation**
7. **Septicemia**

Classification :

	Anterior Epistaxis	Posterior Epistaxis
Incidence	Common	Rare
Site	Little's area or anterior part of Lateral wall	Postero superior part of nasal cavity, often difficult to localise the bleeding point
Age	Mostly occurs in children or young adults	After 40 years
Cause	Usually trauma	Spontaneous, often due to Hypertension or Arteriosclerosis
Bleeding	Usually mild can be easily Controlled by local pressure or anterior pack	Bleeding is severe, requires hospitalisation, postnasal pack often required.

Made to sit upright and incline forwards with mouth open and pinched (self) nostrils, then asked to breathe .

Common sites of bleeding

1. **Little's area** – In most of cases bleeding is from the antero-inferior arterial plexus of septum. The vascular plexus formed at this area is referred as ***"Kiesselbach's plexus"***. (Formed by the anastomosis of Septal branch of sphenopalatine artery, Anterior ethmoidal artery, Greater palatine artery and Superior labial artery).
2. **Middle turbinate** – As it is the dividing line between the Internal and External carotid artery and demarcated by drawing an imaginary line on the septum parallel with turbinate.
3. **From Superior meatus, turbinate** and corresponding septum by a branch of Nasopalatine artery.
4. **Woodruff's plexus** – Large vessels originating from the posterior pharyngeal wall of inferior meatus. These are venous in origin.

Investigations

1. *Vital parameters* – Shock detected
2. *Hemoglobin* – Anemia
3. *Coagulation tests* – Bleeding disorders
4. *Radiology* – Sinusitis, fractures, tumours
5. *Biopsy* – Malignancy

Management :

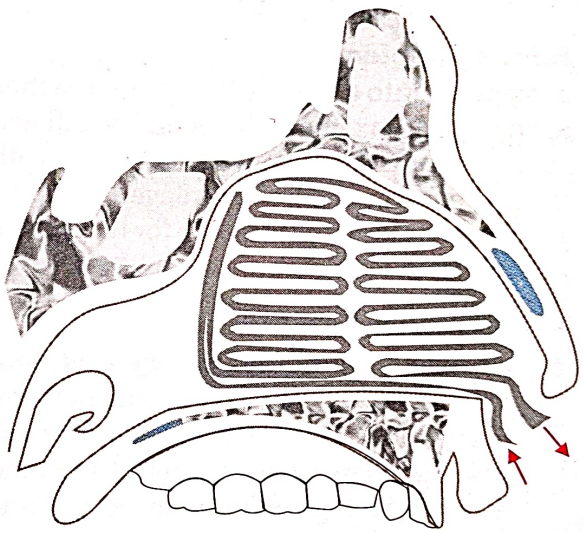
1. ***Immediate***
   a) *Pressure* – By compression of the nose between the thumb and first two fingers.
   b) *Ice cold packs* – Applied on the bridge of the nose, which may stop the bleeding by reflex vasoconstrictions.
   c) *Trotter's method* – The patient is quietly spitting out all the blood from the pharynx.
2. ***Assessment of blood loss*** and vital parameters checked. Treatment of shock and hypovolaemia.
3. ***Measures to control bleeding:***

i. ***Cautery***
   a) *Chemical* – After the nose is anaesthetised by 4% lignocaine and adrenaline, then 10% silver nitrate or 50% trichloroacetic acid is used.
   b) *Electrical* – Done when the bleeding point is visualised.

ii. ***Cryo surgery*** – Is direct application of the probe tip.

iii. ***Packing***

a) Anterior nasal packing –

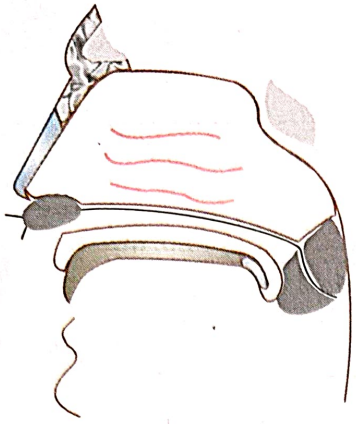


Anterior nasal packing

This is performed in a systematic manner i.e. from anterior to posterior and inferiorly to superiorly, the pack is placed. The nasal pack may be a furacin or iodoform gauze which is removed after 48 hours. If bleeding does not stop with this, a post nasal pack is required.

b) *Post nasal packing*

It is performed by drawing a post nasal pack into the nasopharynx with the help of two rubber catheters through the nostrils. An anterior nasal pack is again required here to keep this pack in place.



Foley's catheter with inflated ballon in the nasopharynx

iv. ***Foley's catheter***

It is passed into one nostril and as it reaches the nasopharynx, the catheter bulb is inflated such that the balloon lies in firm contact with the postnasal area. The advantage being, this procedure is less troublesome for the patient as well as the surgeon.

v. ***Surgery – Ligation of :***

a) ***Anterior ethmoidal artery*** – Is done via an external ethmoidectomy incision. The periosteum is elevated from lacrimal fossa and lamina papyracea of ethmoid, after achieving haemostasis, then retract the medial orbital periosteum and lacrimal sac. The artery seen at the junction of medial and superior walls of the orbit is divided and coagulated. Incision closed.

Just behind Anterior ethmoidal artery, Posterior ethmoidal artery is situated which can be ligated.

b) ***Maxillary artery*** – In the pterygopalatine fossa via Caldwell-Luc operation.

c) ***External carotid artery*** – Via a neck incision.

4. General Measures :

a) Sedation with diazepam or pethidine. This reduces restlessness of the patient, allays anxiety and reduces hypertension.

b) Hematinics and multi-vitamins to improve the hemoglobin.

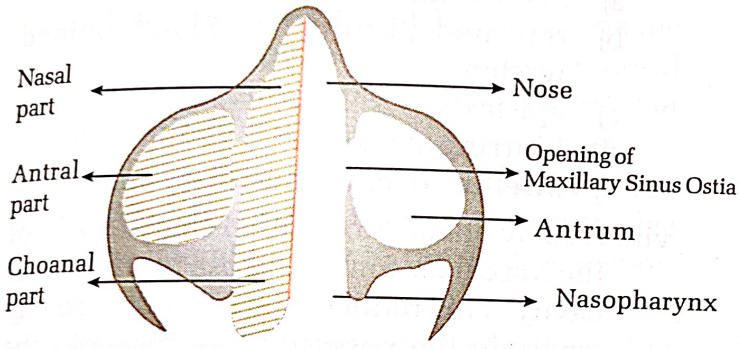
c) Antibiotics are required along with nasal packing to prevent infection.

CHAPTER 29
Nasal Polyps

Polyp is prolapsed edematous respiratory mucosa.

Types:

1. Antrochoanal
2. Ethmoidal



Horizontal section at the level of the Maxillary Sinus ostia

ANTRO-CHOANAL POLYP (Killian's polyp)

An antrochoanal polyps arises from the Maxillary antrum, enters the nasal cavity through the natural ostia of the antrum and also passes backwards into the posterior choanae.

Etiology

i. ***Age*** – common in the 2nd decade.
ii. ***Sex*** – both sexes equally affected.
iii. ***Predisposing factors*** – infection and allergy.

Gross Pathology – "Trifoliate"

Parts :

i. **Antral** – is the first part to form, resulting from edema of the antral mucosa. Once the maxillary antrum is full of the polyp, it finds its outlet at the opening of the ostia in the middle meatus.

ii. ***Choanal*** – part is seen in the nasopharynx. This is usually the largest part of the polyp and its formation is due to :

a) Direction of the opening of the maxillary ostia, which points downwards and backwards.

b) Direction of movement of the cilia towards the nasopharynx aid the formation of this part.

c) Floor of the nasal cavity is sloping backwards.

d) Inspiration creating a greater amount of negative pressure, pulls the polyp backwards.

iii. ***Nasal*** – Usually smallest in size. The tip may undergo discolouration (blackening), which occurs due to metaplasia of the exposed epithelium.

Microscopic Pathology

The polyp is lined by pseudo-stratified columnar epithelium. The nasal part is exposed, therefore undergoes metaplasia causing it to change to stratified squamous epithelium. Eosinophils are found in plenty in the polyp tissue.

Symptoms

1. ***Nasal block*** – Is initially on the side of the polyp and later becomes bilateral when the polyp occupies the nasopharynx.
2. ***Headache*** – Due to blockage of the sinus ostia.
3. ***Nasal discharge*** – May be watery or purulent, if infection present.
4. ***Sneezing*** – Due to allergy.
5. ***Mouth breathing*** – Following complete nasal block.

Signs

1. External nose is normal.
2. Anterior rhinoscopy reveals a single, unilateral polyp.
3. Posterior rhinoscopy shows a polyp in the nasopharynx.

Radiography

1. *Paranasal sinus (Water's view)* – shows an opaque maxillary sinus.
2. *Lateral skull* – would show a soft tissue shadow arising from anteriorly in the nasopharynx.

Treatment

1. Before 16 years – FESS or Polypectomy. (as dentition and mid-face development is not complete).
2. After 16 years – FESS (Functional Endoscopic Sinus Surgery) or Caldwell-Luc Surgery.

CALDWELL – LUC

Definition

It is an opening made on the anterior wall of Maxilla via the canine fossa to visualise and remove disease from the Maxillary sinus. This operation is followed by an intra-nasal antrostomy.

Caldwell in New York and Luc in Paris described the same operation around the same time.

Indications

i. Antrochoanal polyp in patients above the age of 16 years.

ii. Chronic Maxillary Sinusitis not responding to the conservative line of treatment, which means at least 3 antral punctures. (this indicates an irreversible mucosal disease).

iii. Removal of a Dentigerous cyst. The cavity of a dentigerous cyst lies anterior to the Maxilla, yet the antrum is opened. The cyst cavity and the antrum are placed so as to marsupalise the cyst. This allows epithelisation of the cavity.

iv. Elevation of depressed fracture of the Maxilla by packing the cavity.

v. Removal of foreign bodies like bullets, rhinolith or impacted roots of teeth left behind on extraction.

vi. Whitmack's operation – Implantation of the Stenson's duct into the Maxillary Antrum in cases of Atrophic Rhinitis.

vii. To examine and biopsy any tumour involving the Maxilla which is not clinically evident in the nose or oral cavity. Done for symptoms of :
   a) altered smell
   b) repeated purulent or blood stained washes
   c) epistaxis
   d) blurring of vision
   e) swelling over the maxillary area.

viii. Raghav Sharan's operation-Implantation of the Maxillary Sinus mucosa into the nasal cavity. This reduces the size of the roomy cavity in the nose and also increases the mucosal area thus treating Atrophic Rhinitis.

ix. Orbital Decompression. Done only for expothalmus not responding to steroids.

x. Blow out fractures of the orbit. The roof of the Maxillary Sinus is elevated and Sinus packed. Such fractures are caused by a blow or hit with a ball causing the inferior wall of the orbit to give way.

xi. Transantral Ethmoidectomy. Done for ethmoiditis with evidence of ipsilateral maxillary sinusitis, known as Jansen-Horgan operation.

xii. Transantral Vidian Neurectomy.

xiii. Transantral Maxillary nerve section.

xiv. Transantral Maxillary artery ligation.

xv. Removal of benign Maxillary tumours.

xvi. Closure of an Oro-Antral fistula.

xvii. Approach to the Pituitary fossa for hypophysectomy.

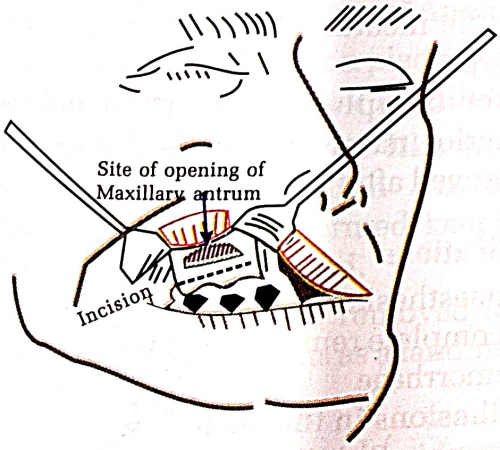
xviii. Implantation of Radioactive needles in cases of malignancy of the Maxilla.

Contraindications

1. Below 16 years of age, as growth of middle third of face and dentition is not complete.
2. Acute Sinusitis, as it causes excessive bleeding and dissemination of infection.
3. Diabetes and hypertension.
4. Bleeding disorders.
5. Any general contraindications to surgery.
6. Recent trauma.

Technique

1. Usually performed under local anaesthesia. 2% lignocaine with 1 in 2 lac adrenaline is injected into the gingivolabial sulcus on the side to be operated on. With General anaesthesia , the throat is packed to prevent any aspiration of blood.
2. Incision is made bone deep 1 cm below the gingivo-labial sulcus from the posterior edge of the lateral incisor to the first or second molar.
3. Elevation of the periosteum to make the anterior wall of maxilla bare upto the infra-orbital foramen superiorly.
4. Anterior wall of the Antrum is opened with a gouge and hammer, the opening having a diameter of 3 cm.
5. The indication for which CWL is performed is carried out.
6. Bleeding is usually minimal and stops with gelfoam pieces. If bleeding persists, then a pack is left in the Maxilla which is removed from an intranasal antrostomy.



Site of opening of the Antram which is above the incision/suture line

Complications

I. *Immediate*

i. Anaesthesia
ii. Damage to teeth
iii. Damage to infra-orbital nerve
iv. Bleeding
v. Damage to orbital floor

II. *Late*

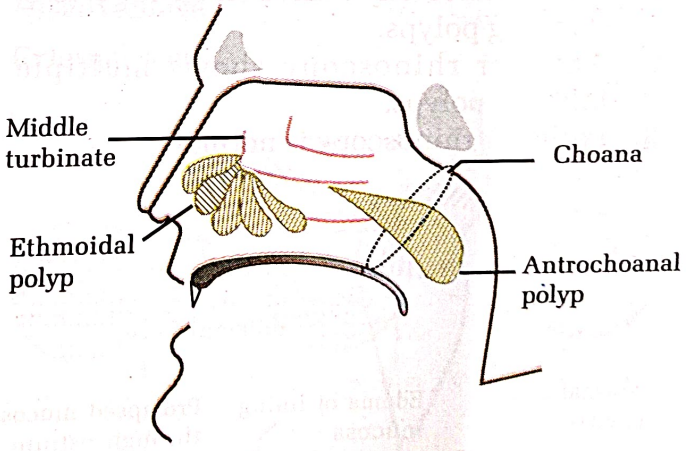
i. Infra-orbital neuralgia
ii. Dental neuralgia
iii. Devitalisation of teeth
iv. Oro-antral fistula
v. Bony deformity

Bleeding from any of the following sites can occur

i. Mucosal vessels at the incision
ii. Anterior superior alveolar vessels.
iii. Infra-orbital vessels
iv. Maxillary artery
v. Sphenopalatine vessels
vi. Ethmoidal vessels

Prognosis

Very good as never undergoes a malignant change. Recurrence of the polyp is rare, if a Caldwell-Luc operation is done.



Nasal Polypi

ETHMOIDAL POLYP

Ethmoidal polyp arise from the mucosa of the ethmoidal air cells and by the effect of gravity occupy the anterior choanae.

Etiology

i. ***Age*** – Mostly seen in the elderly
ii. ***Sex*** – Both are equally affected
iii. ***Predisposing factor*** – Allergy
iv. ***Bernoulli's Phenomenon*** – When air passes through an area of constriction, it creates a negative pressure around which causes increased tissue fluids accumulation. This leads to a polyp formation.

Gross Pathology

Bilateral multiple polyps, hanging downwards.

Microscopic Pathology

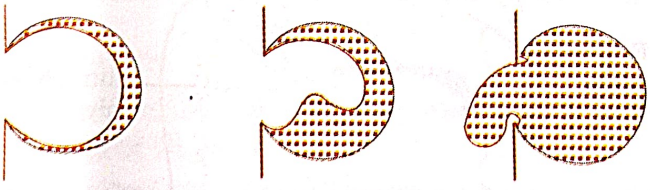
Mucous membrane is lined by pseuo-stratified squamous epithelium. Submucosal tissue is edematous with intercellular fluid content.

Symptoms

1. Nasal block
2. Sneezing
3. Headache
4. Nasal discharge
5. Mouth breathing

Signs

1. External nose may be broadened due to long standing polyps.
2. Anterior rhinoscopy shows multiple bilateral polyps.
3. Posterior rhinoscopy is normal.



Normal sinus cavity | Edema of lining mucosa | Prolapsed mucosa through ostium

Ethmoidal polyp formation

Radiography

Caldwell's view of the paranasal sinuses show an opacity of the ethmoidal sinuses as well the nasal cavity.

MANAGEMENT

The protocol of treatment for polypus is

1. Antihistaminics and nasal decongestants, failing which a course of steroids is given after excluding hypertension, coronary artery disease, diabetes, tuberculosis, peptic ulcer or pregnancy in the patients.
2. With no response to medical line of treatment the patient is subjected to a Nasal polypectomy. Ethmoidal polypi are known for its recurrences, as the etiological factor is allergy. A nasal polypectomy is done on 3 occasions for a recurrence.
3. Inspite of polypectomies if a recurrence occurs, an intra-nasal ethmoidectomy is performed, where by 80% of the air cells can be removed.
4. Even then, if a recurrence of polypi occurs, an external ethmoidectomy is done. This is the only method of complete cure from this condition.

Nasal Polyectomy

1. Preferably done under General Anaesthesia. The throat is packed firmly to avoid aspiration of blood.
2. The nose is packed with saline – adrenaline (1 in 2 lac dilution) to minimise the bleeding.
3. Only with a few polypi, a snare can be used where the wire retracts not fully into the sheath and the polypi is avulsed.
4. With large number of polypi, Luc's or Henckel's forceps are used and Killian's long bladed speculum is introduced into the middle meatus to get direct access to the polypi.
5. After complete removal of polypi, an anterior nasal packing is done, which is removed after 48 hours.

Complications

1. Anaesthesia
2. Incomplete removal of polypi
3. Hemorrhage
4. Adhesions in the nose
5. Atrophic Rhinitis.

INTRANASAL ETHMOIDECTOMY

This is a blind procedure and the ethmoids being close to many important structures, it is necesary to have knowledge of the anatomical landmarks.

Surgical Anatomy

1. Natural orifice of the Sphenoid Sinus is situated at 7 cm from the anterior nasal spine. Therefore, ethmoidectomy is complete on reaching the 7 cm mark the instrument.
2. Roof of the sphenoid sinus is continuous with the roof of the ethmoids. Any violation of this limit leads into the anterior cranial fossa.
3. Lateral wall of sphenoid is continuous with the ethmoid wall (lamina papyracea). It is a guide to avoid damage to the orbital contents.
4. Superior attachment of middle turbinate is the medial limit of the ethmoids. Any manipulation crossing this limit has the danger of damaging the Cribriform plate.
5. Nasofrontal duct enters the middle meatus at its antero-superior limit.
6. Nasolacrimal duct enters the inferior meatus at its antero-superior limit.
7. Inferior turbinate is a separate bone (not from ethmoids) and is attached to the lateral wall of the nose.

Technique

1. Preferably done under General Anaesthesia.
2. Nose is packed with cotton pledgets soaked in saline adrenaline (1 in 2 lac dilution).
3. The polypi are removed like in nasal polypectomy and then intranasal ethmoidectomy is performed keeping its anatomic position in mind.
4. The middle turbinate is removed last, as throughout surgery it acts as a landmark for the cribriform plate.

Disadvantages:

1. Only 80% of the cells are removed.
2. The Agger nasi cells cannot be reached.
3. Being a blind procedure, chances of damage to neighbouring structures is high.
4. Recurrence of polypi.

EXTERNAL ETHMOIDECTOMY

Indications

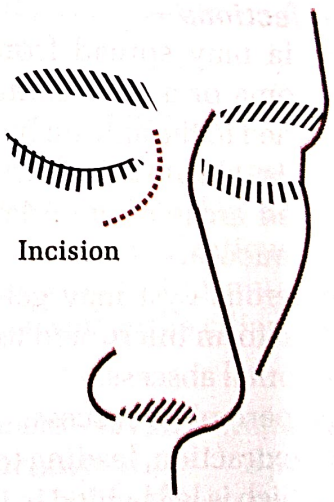
1. Recurrence of Polypi after an intranasal ethmoidectomy
2. Chronic Ethmoiditis
3. Mucocele or Pyocoele of the Ethmoids
4. Epistaxis
5. Approach for
   i. Dacro-cysto-rhinostomy
   ii. CSF leak repair
   iii. Optic Nerve decompression
   iv. Cranio-Facial resection
   v. Sphenoid Sinus
   vi. Pituitary Gland.

Advantages

1. Complete removal of ethmoidal air-cells.
2. No damage to neighbouring structures as done under vision.

Disadvantage

External scar.



Site of incision for an external ethmoidectomy

CHAPTER 30

Sinusitis

Inflammation of the paranasal sinuses is quite common. Amongst all the paranasal sinuses, the maxillary sinuses are most frequently affected.

i. As it lies in close proximity to teeth, and
ii. Floor of the maxillary sinus is at much lower level as compared to its ostia, so normal drainage is also affected.

Acute sinusitis is a condition requiring medical treatment, whereas chronic sinusitis requires surgery.

Etiology of acute sinusitis:

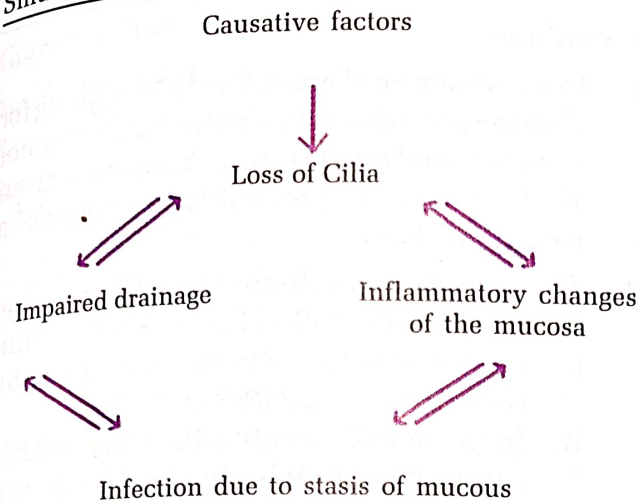
1. **Age** – affects children above 5 years of age but infants are liable to suffer from acute ethmoiditis.
2. **Sex** – affects both equally.
3. ***Acute rhinitis*** – causing spread of infection to the sinuses by way of their natural ostia from the nasal cavities.
4. ***Pharyngeal infections*** – especially tonsillitis in children act as a focus of infection.
5. ***Dental infections*** –
   i. Bacteria may spread from an apical granuloma or a periodontal pocket or are carried to the antrum by lymphatics.
   ii. Chronic dental infections cause localised areas of granulations in the sinus mucosa.
   iii. Dentigerous cyst may get infected in relation to an unerupted tooth.
   iv. Periodontal abscess.
   v. Acute periapical abscess.
   vi. Tooth extraction, leading to a fractured root which is left behind in the maxillary sinus.
   vii. Dental fillings may be driven into the maxillary sinus.
6. ***Swimming and diving*** – lead to water penetrating into the sinus.
7. ***Trauma*** – may be
   i. Contusion of the sinuses
   ii. Compound fracture of one of the sinuses.
   iii. Foreign bodies.
   iv. Barotrauma.
8. ***General Health*** – if poor, precipitates sinusitis.

Predisposing factors:

1. ***Prolonged exposure*** – to cold or even excessive heat, smoke, dust or even overcrowding.
2. ***Obstruction :***
   i. *Anatomical* – which is caused by a septal deviation on one side with turbinate hypertrophy on the other side, both causing blockage of the ostia of the paranasal sinuses.
   ii. *Infective* – causing congestion leading into obstruction of the sinus ostia.
   iii. *Allergy* – causing mucosal swelling or even polyps.
   iv. *Tumours* – usually are malignant.
3. ***Chest conditions***
   i. Bronchiectasis
   ii. Bronchitis
   iii. Asthma.

Organisms

1. ***Viral*** – rhinovirus, parainfluenza virus, respiratory synctial virus.
2. ***Bacteria*** – pneumococci, staphylococcus, streptococcus, E. coli etc.
3. ***Fungi*** – aspergillosis.
4. ***Tuberculosis, leprosy and syphillis*** – rare.



Causative factors and pathophysiology of chronic sinusitis

Pathology:

1. Initial response to infection results into edema and congestion of the sinus mucosa.
2. Excess secretion of mucus occurs , that fills up the sinuses.
3. The cilia get damaged and the ostia blocked, which causes secondary infection of the collected secretion.
4. Depending upon the host resistance, antibiotics used and virulence of the organism, the disease clears up or may progress towards complications.

Symptoms

1. Headache, malaise and fever.
2. Loss of normal vocal resonance
3. Pain around the various sinuses.
4. Referred pain in the ear
5. Nasal discharge
6. Nose block
7. Epistaxis.

Signs

1. Flushing of the cheek with swelling in case of maxillary sinusitis and involving the upper eye lid in frontal sinusitis.
2. Tenderness is elicited at the canine fossa in maxillary sinusitis and above the medial canthus in frontal sinusitis.
3. Anterior rhinoscopy reveals redness and mucosal congestion. Pus may be seen trickling from the middle meatus area.
4. Posterior rhinoscopy may also reveal pus from the middle meatus area.

Investigations

1. *Bacteriological* – examination is required if the sinusitis does not respond to antibiotic treatment.
2. *Radiology* – Water's and Caldwell's view of the paranasal sinuses may show
   i. Mucosal thickening
   ii. Opacity of sinuses or
   iii. An air-fluid level
3. Diagnostic Antral puncture
4. Nasal sinus endoscopy.

Differential diagnosis

1. Dental neuralgia
2. Temporo – mandibular neuralgia
3. Trigeminal neuralgia
4. Migrane
5. Atypical facial pain
6. Temporal arteritis
7. Cerebropontine angle tumours
8. Nasopharyngeal tumours
9. Insect bite
10. Multiple Sclerosis
11. Angionerurotic edema
12. Malignancy of the sinus
13. Meningitis.

Treatment of Acute Sinusitis:

I. ***Prevention*** – by improving general condition of the patient, if affected by a chronic illness by physical or mental fatigue. Lowering of resistance affects the mucosal blanket integrity as well as the ciliary action.

II. ***Medical***

   i. *Antibiotics* – Erythromycin, Amoxycillin, Azithromycin, Sparfloxicillin Gatifloxicillin etc. can be given

   ii. *Local decongestants* – are used, which do not interfere with the ciliary functions i.e ephedrine or xylometazoline nose drops or sprays are used.

iii. *Analgesics and anti* - inflammatory drugs -paracetamol, diclofenac, nimesulide etc. are commomly used for relief of headache and pain.

iv. *Antihistamics* - terfenadine, chlorphenaramine, loratidine, cetrizine or fexofenadine are used.

v. *Steam inhalation* – for clearing out the secretions. Tincture benzoin or menthol provide symptomatic relief and promote sinus drainage.

III. Surgical

After the active phase is over

1. Correction of a deviated septum
2. Antral puncture.
3. Functional endoscopic sinus surgery.

ANTRAL PUNCTURE

Definition

It is a procedure whereby drainage and washes are given to the Maxillary Sinus by a cannula introduced through the inferior meatus.

Indications:

A. Diagnostic

i. To confirm clinical and radiological diagnosis of chronic Maxillary Sinusitis.

ii. To obtain pus, which is sent for smear, culture and antibiotic sensitivity.

B. Therapeutic

i. Antral lavage for chronic Maxillary Sinusitis.

ii. Antral lavage for Atrophic Rhinitis.

iii. Instillation of medicaments through the cannula.

Contraindications

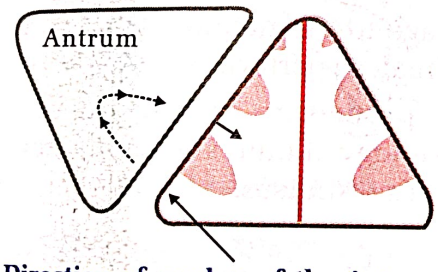
1. Not performed in children less than 7 years of age, as the Maxillary Sinus is not fully developed.
2. Acute Sinusitis
3. Diabetes
4. Bleeding disorders
5. Fracture of the Maxilla.

Technique

1. Done under local anaesthesia, excepting for children or where the antral wall can not be penetrated by the trocar. Atropine is given 1/2 hr. before, to prevent the chances of a vasovagal attack.
2. Three swab sticks dipped in 4% lignocaine are placed at the following sites for 10 min.
   i. In the inferior meatus to block the superior alveolar nerve.
   ii. In the middle meatus, the swab stick is pushed posteriorly to block the sphenopalatine ganglion and its branches.
   iii. In the roof of the nose to block the anterior ethmoidal nerve.
3. The Tilley-Lichwitz trocar cannula is introduced into the inferior meatus, in a direction pointing towards the medial canthus of the eye.
4. On meeting with resistance at the upper end of the inferior meatus, the direction of cannula is changed towards the lateral canthus of the eye.
5. In a screwing action, the trocar directly enters into the maxillary sinus. There is a sudden feeling of give way to the resistance and the instrument hits the lateral wall of the antrum.
6. The trocar is totally withdrawn and cannula moved in various directions, to confirm its postion in the antrum.
7. A Higginson's syringe is attached to the cannula at one end and other being in a bowl of normal saline. The patient is asked to bend forwards and breathing is with mouth open.
8. The washings are continued till the returning fluid is of the same colour as normal saline is pumped in.
9. The cannula is removed and nose packed for 10 min with a cotton pledget to prevent bleeding.
10. The patient is given antibiotics and nasal decongestants. Antral puncture under local anaesthesia is done as an OPD procedure.

Complications

1. Anaesthesia
2. Vasovagal shock
3. No return of fluid, may be due to
   i. Blocked cannula
   ii. Cannula abutted against the lateral wall of the antrum.
   iii. Ostia of Maxillary Sinus may be blocked. Here we introduce another trocar cannula, keeping the first in place.
   iv. False passage created, that is the cannula may be in the cheek tissues.
4. Proptosis caused by damage to the orbital floor.
5. Bleeding from local veins.
6. Air embolism due to an injured vein during the procedure. Therefore no air is pushed into the antrum once the saline is over.
7. Introduction of infection into the Maxillary Sinus, if aseptic precautions not taken.



Direction of washes of the Antrum

Puncture Sites

1. The normal ostia of the Maxillary Sinus.
   However, this is not used as
   i. Reaching the site is not easy
   ii. Manipulation around the normal ostia would lead to edema and fibrosis, causing further stenosis of the ostia leading to aggravation of symptoms.
   iii. Greater chances of damaging neighbouring structures, i.e. Orbit Cribriform plate etc.
2. Canine fossa. This is not used as the chances of osteomyelitis are higher.
3. Puncture via the Inferior meatus is preferred, due to :
   i. Easy accessibility
   ii. Chances of damaging important structures is remote.
   iii. Medial wall of Maxilla is formed by compact bone, so osteomyelitis does not occur.

Antral Puncture in Atrophic Rhinitis

1. The Antrum is sclerosed, therefore bone is hard and penetration difficult.
2. Landmarks are lost, as crusts and pus are already present in the nose.
3. The cannula does not remain steady as the inferior turbinate is atrophied which normally supports the cannula.

Complications of Acute Sinusitis:

1. Chronic sinusitis
2. Pansinusitis
3. Otitis media
4. Pharyngitis and laryngitis
5. Mucocoele and Pyococele
6. Osteomyelitis
7. Asthma
8. Cavernous sinus thrombosis
9. Orbital cellulitis
10. Orbital abscess
11. Superior orbital fissure syndrome.
12. Meningitis
13. Cavernous sinus thrombosis
14. Brain abscess-frontal lobe
15. Oro-antral fistula
16. Dental abscess
17. Nasal polyposis
18. Secondary atrophic rhinitis
19. Atelectasis
20. Tonsillitis.

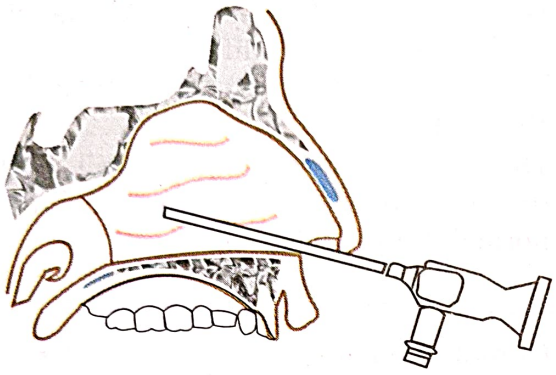
Treatment of Chronic Sinusitis:

i. Functional Endoscopic Sinus Surgery
ii. Intranasal Antrostomy
iii. Caldwell-Luc.

I. FUNCTIONAL ENDOSCOPIC SINUS SURGERY

It is visualisation and clearance of disease from the nose and paranasal sinuses, also referred to as FESS (Functional Endoscopic Sinus Surgery). The middle meatus is the site of opening of the various sinus ostia and thus this

area is critical in causing sinusitis (osteo-meatal complex). When done with the help of Diode LASER it is called Functional Endoscopic Endonasal Laser Surgery (FEELS).



Rhinoscopy using an endoscope

Indications:

A) Diagnostic

i. Chronic sinusitis
ii. Unexplained headache
iii. Idiopathic epistaxis
iv. Assessing eustachian tube function
v. Velopharyngeal incompetance
vi. Nasal foreign body.

B) Therapeutic

i. Chronic sinusitis
ii. Antrochoanal and ethmoidal polypi removal
iii. Dacrocystorhinostomy (DCR)
iv. Foreign body removal.

Muco-ciliary clearance is directed towards the natural ostia

Principle

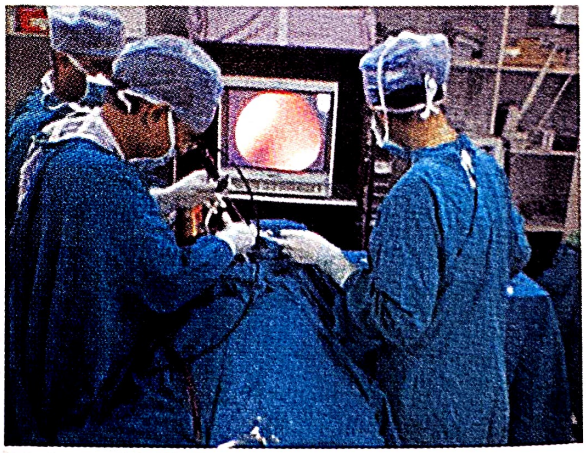
Drainage of the paranasal sinuses take place from their normal ostia, as the ciliary action is directed towards the same. In nasal sinus endoscopy the diseased or blocked ostia are opened and widened thus facilitating mucosal clearance with the help of the normal ciliary mechanism. This is physiological procedure, as in accordance with normal mucociliary clearance pathways.

Complications

1. Anaesthesia
2. Hemorrhage
3. Nasal adhesions
4. Orbital damage
5. Optic nerve damage
6. CSF rhinorrhea
7. Atrophic rhinitis.

Advantages:

1. Performed under local anaesthesia except in children where general anaesthesia is given.
2. Damage to the neighbouring structures is minimal, as performed under direct vision.
3. This procedure confers to the normal physiology of mucociliary clearance of the paranasal sinuses.



Nasal Sinus endoscopy setup

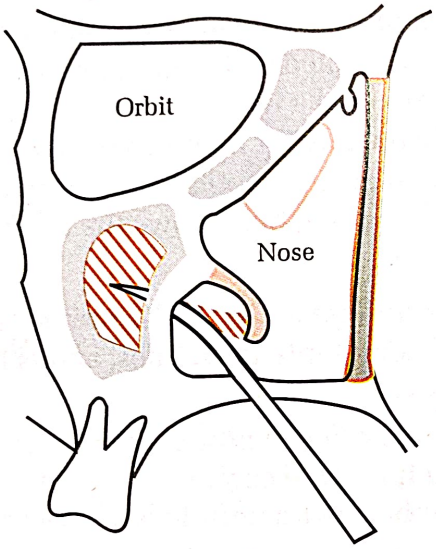
II. INTRA NASAL ANTROSTOMY

Definition

It is a permanent opening made in the medial wall of the Maxillary antrum for purpose of drainage and washes of the antrum.

Indications

1. Chronic Maxillary Sinusitis, not responding to repeated antral punctures.
2. Biopsy in a suspected case Malignancy of the Maxilla.
3. As a part of Caldwell-Luc operations.
4. Elevation of depressed fracture of the Maxilla.
5. Where Sinus Endoscopy is to be performed repeatedly.



Intranasal Antrostomy

Technique

1. Performed under local anaesthesia.
2. The Tilley's antrostomy harpoon is placed in the inferior meatus to create an opening.
3. This opening is widened and edges smoothened with an antral burr.
4. Washes are given using an antral washing cannula.

Disadvantages:

1. Normal mucociliary clearance takes place from the normal sinus ostia, so opening at another site is of no use.
2. Inhalation of cold air blast may cause pain in the sinus.
3. On bending the head down, mucus plugs may fall out of the sinus.

III. Caldwell-Luc. (page 126)

CHAPTER 31
Tumours of the Nose and Paranasal Sinuses

Types :

I. *Benign*

A) Epithelial
- i. Papilloma
- ii. Adenoma

B) Connective tissue
- i. Fibroma
- ii. Osteoma
  - a) Cancellous osteoma
  - b) Fibrous dysplasia
  - c) Compact osteoma
- iii. Angioma
  - a) Capillary
  - b) Cavernous
  - c) Multiple telangiectasis
- iv. Osteoclastoma
- v. Cysts
  - a) Congenital cysts
  - b) Cystic odontomes
  - c) Mucoceles
  - d) Cysts of bone
  - e) Dermoids

Multiple Telangiectasis

Multiple Telangiectasis (Oslers disease) may occur in the nose in the Little's area, and may affect simultaneously the face ,tongue and lips. It may be familial.

Management :

1. LASER is the modality of choice.
2. Ethinyloestradiol given in the dose of 0.5 mg daily may help
3. Excision followed by skin grafting is advised in recurrent cases (Septodermoplasty)
4. Irradiation is sometimes recommended.

Cystic Odontomes

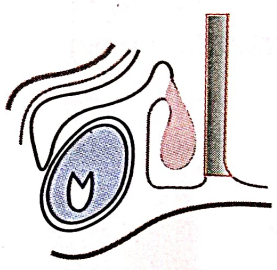
A. ***Cyst of eruption:*** A bluish swelling may be seen over an unerupted deciduous or permanent tooth

B. ***Dental cyst :*** occurs at the apex of a carious tooth arising from the epithelial remains of the peri-odontal membrane.The infected tooth produces apical granuloma which forms the cyst.

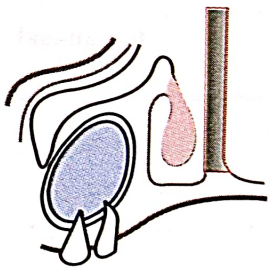
C. ***Dentigerous cyst :*** arises from the follicle of an unerupted tooth, and contains a tooth. It presents as a firm swelling in the cheek, inside the nose or in the bony palate. It can be identified by noticing a missing tooth, and the radiograph shows the tooth in the cyst.

D. ***Adamantinoma (Ameloblastoma) :*** occurs more commonly in females, and is locally invasive.

A diffuse swelling is produced with egg-shell crackling.It is more common in the mandible. Radiograph shows a multiloculated swelling.



Dentigerous cyst containing a tooth



Dental cyst secondary to a caries tooth

Dentigerous cyst	Dental cyst
1. Seen in children	Seen in adults
2. Tooth within the cysts	Cyst outside the tooth
3. Cyst in sterile	Cyst is infected
4. Caldwell-Luc done	Treated by dental (extraction)

Management

All the odontomes can be excised.Dental and dentigerious cysts are removed via a sublabial incision. Adamantinoma requires a radical excision.

Mucocele

It is a cystic swelling of the sinus secondary to blockage of the sinus ostium (due to blockage of a mucous gland or obstruction of the fronto nasal duct) resulting in thinning and expansion of the sinus wall.

It commonly occurs in the frontal sinus or sometimes ethmoids may be affected.There is a swelling above the inner canthus of the eye and contains sterile fluid.The swelling is bony initially, but as the bony wall breaks down, it prolapses to the exterior. Proptosis may be caused by its prolapse into the orbit. A pyocele may form, if it gets infected.

Radiographs show enlargement of the sinus with haziness and rounding of the borders.

Management :

Excision by external incision is performed. Re-establishment of frontonasal duct drainage is done by leaving a drainage tube from frontal sinus to nasal cavity for frontoethmoid mucocele (Howarth's operation).

II. Malignant

A) Epithelial

i. Carcinoma
   a) Squamous cell carcinoma
   b) Adeno carcinoma
ii. Mixed salivary tumours

B) Connective tissue

i. Sarcoma
ii. Melanoma

C) Metastatic tumours – rare

MALIGNANCY OF THE MAXILLARY SINUS

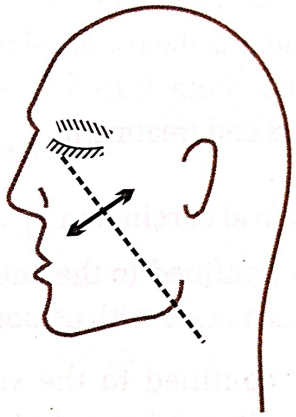
It is the commonest of the tumours of the nose and paranasal sinuses.

Etiology

1. ***Age*** – seen in the elderly, usually after the age of 50 years.
2. ***Sex*** – males are more frequently affected.
3. ***Smoking*** – increases the incidence.
4. ***Wood workers*** – show a high incidence of adenocarinoma.
5. ***Snuff*** – has been blamed for it.
6. ***Chronic sinusitis*** – is an unlikely precursor of cancer.

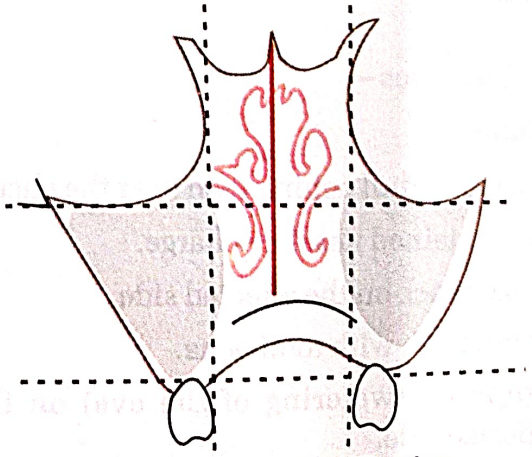
1. Anatomical classification

A) Onhgren's line – is an imaginary line passing from the inner canthus to the angle of the mandible, tumours situated above this line have worse prognosis than those situated antero – inferiorly (antro-alveolar).



Onhgren's Line

B) Lederman's lines - (divide the face into various quadrants by vertical and horizontal lines). One horizontal line passes through the floor of orbit, other line passes through the floor of maxillary antrum, where as vertical lines passes through the inner canthus of the eyes. Tumours involving more than one quadrant have worse prognosis.



Lederman's classification

2. Morphological classification

i) Primary tumours – squamous cell carcinoma or adenocarcinoma

ii) Secondary (metastasis) – from breast, prostate, parotid etc

3. TNM\AJC classification :

American Joint Committee – has also taken Ohngren's plane into consideration to divide antroethmoidal growths into suprastructural (above the plane) and infrastructural (below the plane) and further classified them into T.N.M. system for prognosis and treatment.

Staging of antral carcinoma

T_1 Tumour confined to the antral mucosa of the infrastructure with no bone destruction

T_2 Tumour confined to the suprastructure mucosa without bone destruction or the infrastructure with destruction of the medial or inferior walls only.

T_3 More extensive tumour invading the cheek, orbit, anterior ethmoidal air cells, or pterygoid muscles

T_4 Massive tumour invasion of cribriform plate,posterior ethmoid air cells,sphenoid sinus, nasopharynx, Pterygoid plate or base skull

Clinical features –

Symptoms

1. Persistant dull aching pain over the cheek.
2. Blood stained nasal discharge.
3. Nasal block on the affected side.
4. Loose teeth with toothache.
5. Epiphora (watering of the eye) on the affected side.

Spread:

1. ***Anterior*** – cheek leading to swelling
2. ***Posterior*** – pterygoid fossa may cause trismus.
3. ***Superior*** – orbit causing diplopia with proptosis of the eyeball
4. ***Inferior*** – palate and oral cavity leading to falling of teeth
5. ***Medial*** – nasal cavity causing swelling, block, parosmia and anosmia.

The symptoms are dependent upon the involvement of these above structures.

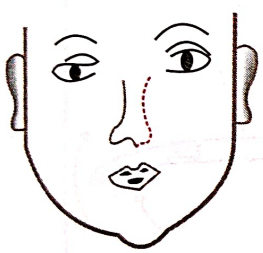
Signs:

1. ***Cheek*** – may be swollen and there may be anaesthesia.
2. ***Anterior rhinoscopy*** – may reveal a nasal mass.
3. ***Posterior rhinoscopy*** – may reveal spread to the nasopharynx.
4. ***Examination of the eye*** – for checking vision and eye ball movement.
5. ***Cranial nerve testing***
6. ***Neck examination*** – for lymph node metastasis (submandibular lymph node).

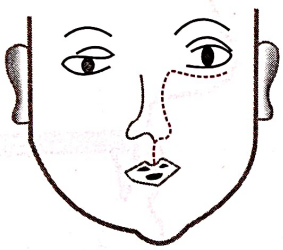
Investigations:

1. ***Water's view*** – would show an opacity, as well as destruction of the sinus walls.

2. ***Biopsy*** – from a nasal mass. An Intranasal antrostomy approach is preferred, because if diagnosed as a malignant tumour.
   i. Surgical treatment would involve removal of the lateral wall of the nose (maxillectomy) and thus prevent seeding of tumour cells, which would occur after a Caldwell-Luc.
   ii. If radiotherapy is given, the intranasal antrosomy provides drainage to the desquamated cells.
3. ***CT Scan*** – is mandatory to have knowledge of the exact size and extent of spread of the tumour.
4. ***Antral puncture*** – washings may detect the malignant cells on cytological examination.
5. ***Nasal sinus endoscopy with biopsy.***



Lateral Rhinotomy



Weber-Ferguson's incision

Differential diagnosis:

As for maxillary sinusitis.

Treatment

I. Surgical

1. Partial maxillectomy
2. Total maxillectomy
3. Radical maxillectomy.

Inoperable due to:

1. Nasopharyngeal involvement
2. Intracranial spread
3. Infratemporal fossa involvement
4. Horner's syndrome
5. Bilateral spread to ethmoids or orbit
6. Poor general condition of the patient
7. Distant metastasis.

II. Radiotherapy

6000 Rads are given over a 6 weeks period. 200 rads are given daily for 5 days a week, totalling to 1000 rads per week.

III. Chemotherapy

1. Results are short lived
2. Very expensive
3. Side effects require constant monitoring of hepatic, cardiac, renal pulmonary and hematological functions.

CHAPTER 32

Rhinolalia

Rhinolalia is nasal speech or a change in the quality of voice due to nasal or nasopharyngeal problems.

Types:

I. ***RHINOLALIA CLAUSA*** – where the voice becomes flat as a result of space occuping lesions in the nose or nasopharynx, which prevent the normal escape of air into the nose. This prevents the normal resonance of voice.

Causes:

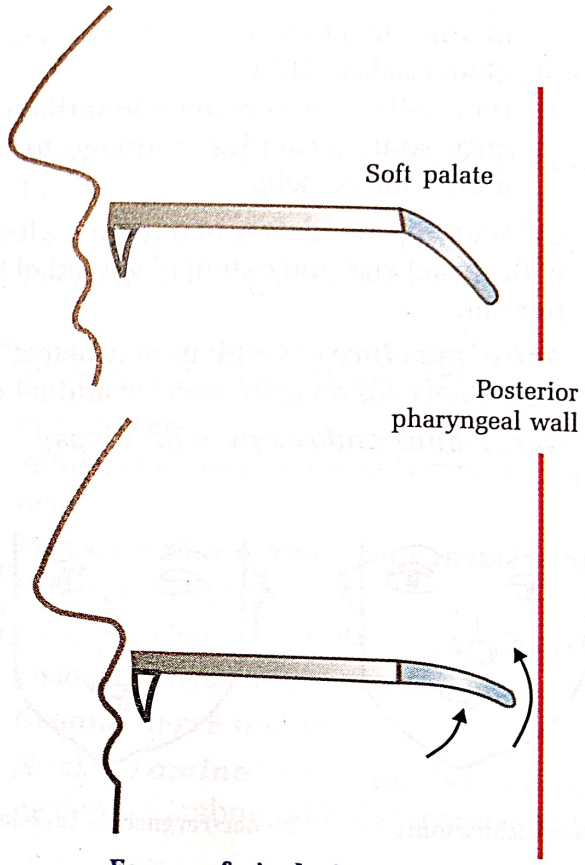
1. Nasal polyps
2. Deviated nasal septum
3. Tumours
4. Adenoids
5. Hypertrophied turbinates
6. Nasopharyngeal angiofibroma

Management – includes treatment of the condition which is causing an obstruction to the normal passage of air during speech.

II. **RHINOLALIA APERTA** – Occurs following inadequate closure of the nasopharyngeal sphincter during speech. This leads to a constant escape of air into the nose causing a nasal twang of speech.

Causes:

1. Post-adenoidectomy
2. Cleft palate
3. Submucous cleft
4. Palatal perforation
5. Removal of angiofibroma
6. Palatal cicatrisation.
7. Palatal palsy.



Escape of air during speech

Management:

1. ***Speech therapy*** – to achieve a closure of the nasopharyngeal sphincter during phonation. Useful in mild cases.
2. ***Surgery*** – performed when conservative treatment fails or a major defect is present. The various forms of surgery are :
   i. Push-back of the palate by a V-Y plasty
   ii. Pharyngoplasty
   iii. Implantation of inert materials in the posterior pharyngeal wall e.g. silastic, teflon etc.
   iv. Pharyngeal flaps to decrease the size of the sphincter.

In cases of cleft palate, one should always avoid Adenoidectomy. Adenoidectomy causes escape of air, increasing the nasal twang.

Causes

HYPONASALITY

Adenoids
Nasal allergy
Angiofibroma
Common cold
Familial
Ethmoidal polyps
Deviated septum

HYPERNASALITY

Habitual
Submucous cleft palate
Post adenoidectomy
Oro-nasal fistula
Paralysis of palate
Velo-pharyngeal insufficiency.

CHAPTER 33
Miscellaneous

FOREIGN BODIES IN THE NOSE

Mode of entry:

1. Anterior nares – most common
2. Posterior nares – during coughing, vomiting and regurgitation e.g. round worm
3. Penetrating wounds and nasal surgery
4. Palatal perforation
5. Calcification in situ i.e. rhinolith
6. Sequestration of bone in situ.

Age

1. Children form the majority of the patients.
2. Adults may be mentally disturbed patients or having undergone surgery or being victims of bullet injuries.

Types of foreign bodies:

1. ***Animate*** – maggots, worms etc.
2. ***Inanimate*** –
   i. Vegetable foreign bodies – peas, beans, nuts etc.
   ii. Matter – beads, pebbles, screws, buttons, paper etc.
   iii. Following surgery – swabs, bone, cartilage etc.
   iv. Sequestra – in syphilis, neoplasm, trauma etc.
   v. Rhinolith.

Pathology

Most of the foreign bodies are inert and may remain in the nose for years leading to mucopurulent discharge and epistaxis.

Clinical features

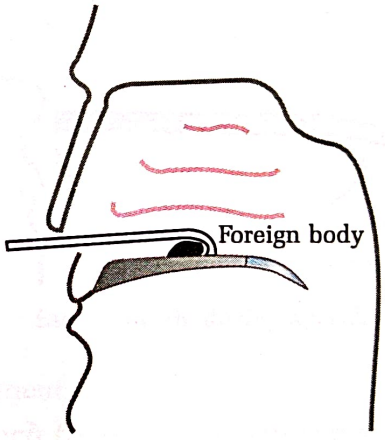
1. Unilateral nasal discharge, which is foul smelling with vegetable foreign bodies.
2. Nasal obstruction and headache
3. Epistaxis
4. Sneezing.

Diagnosis

1. Usually apparent on inspection
2. Nasal endoscopy would clear doubts regarding the presence of a foreign body
3. Radio-opaque foreign bodies are detected on X-rays of the nose.

Management

1. Animate foreign bodies are treated with nose drops (liquid paraffin) and then removed as they get killed due to suffocation.
2. Inanimate foreign bodies are removed using a curved instrument i.e. eustachian catheter.



Removal of nasal foreign body

3. General anaesthesia may be required if
   i. Patient is apprehensive and uncooperative.
   ii. Bleeding is anticipated because of granulations.
   iii. Risk of pushing the foreign body into the nasopharynx.
   iv. Extensive examination of the nose is also required.

RHINOLITH

Rhinolith is a concretion formed around a foreign body, blood or mucus.

Etiology:

1. ***Exogenous*** – Foreign body is the commonest cause.
2. ***Endogenous*** – Blood clot or inspissated pus may be surrounded by concretions.

Pathology

Rhinolith may be single or multiple, friable or hard. The concretion is formed of phosphates, carbonates, calcium and magnesium arranged in concentric layers.

Clinical features

Preferably removed under general anaesthesia. It may be necessary to break a large rhinolith, within the nose for its easy removal. Very rarely is a lateral rhinotomy or a Caldwell-Luc is required.

MYIASES (MAGGOTS IN THE NOSE)

Myiases is an infestation of the nasal cavities by maggots which are larvae of a fly (Genus Chryosomia).

Etiology

1. Poor hygenic condition
2. Atrophic rhinitis
3. Chronic sinusitis.

Pathology

The flies lay eggs on nasal discharge and crusts. The eggs may also be deposited in a slight abrasion or crack in the mucous membrane.

Clinical features

1. Maggots, seen on anterior rhinoscopy
2. Foetid nasal discharge
3. Epistaxis
4. Fever
5. Swelling around nose and eyes.
6. Nasal deformity due to destroyed cartilage.

Complications

1. Spread to neighbouring structures
2. Septicemia
3. Suicide.

Treatment

1. Antibiotics and anti-inflammatory agents.
2. Liquid paraffin or olive oil drops cause the maggots to crawl out of the nose.
3. Turpentine packs irritate the maggots and thus bring them out.
4. Physical removal of the maggots with nasal dressing forceps.
5. Nasal douches to prevent crusting.

CSF RHINORRHOEA

It is the flow of cerebrospinal fluid from the nose.

Etiology

1. ***Traumatic*** – following
   i. Septal surgery
   ii. Intranasal ethmoidectomy and nasal sinus endoscopy (FESS).
   iii. Head injuries.
2. ***Spontaneous***
   From destructive lesions involving the floor of anterior cranial fossa
3. ***Congenital dehiscence***
4. ***Indirect***
   A leak from the middle ear or mastoid in which the CSF presents as a rhinorrhoea coming via the eustachian tube.

Sites of leakage

CSF from anterior cranial fossa reaches the nose by way of cribriform plate, ethmoid air cells or frontal sinus. CSF from middle cranial fossa reaches the nose via sphenoid sinus.Sometimes injuries of temporal bone result in leakage of CSF into the middle ear and hence via the eustachian tube into the nose (otorhinorrhoea).

Clinical features:

1. Rhinorrhoea-clear,watery,increases on coughing or straining.
2. Headache-due to a fluctuation in the CSF pressure.
3. Meningitis-past history.

Differential diagnosis:

1. ***Nasal mucus*** – causes crusting in the nose and will dry up on a handkerchief, while CSF does not.
2. ***Lacrimal secretion*** – is usually of less quantity.

Investigations:

1. ***X-rays*** – may show a fracture line.
2. ***Secretions*** – are examined for glucose and chlorides.
3. ***Handkerchef*** – test is done. Mucous discharge would dry and make the cloth stiff, while CSF would not stiffen the cloth.
4. ***Nasal endoscopy*** – may locate the site of leak.
5. ***Contrast studies*** – It is done by intrathecal injection of a dye (fluorescein 5%, 1 ml) or a radioisotope and placing pledgets of cotton in the olfactory slit, middle meatus, sphenoethmoidal recess and near the eustachian tube, examining the pledgets for dye or radioactivity.

Olfactory slit	- Cribriform plate
Middle meatus	- Frontal or ethmoid sinus
Sphenoethmoidal recess	- Sphenoid sinus
Eustachain tube	- Temporal bone

6. ***CT scan*** with metrizamide contrast is helpful in localising the site of leak.

Management:

1. Complete bed rest
2. Head high position
3. Antibiotics
4. No forcible nose blowing
5. Avoid nose drops or nasal packing to prevent meningitis.
6. Surgical intervention required only when
   i. The CSF leak does not decrease with the above management, even after a period of 6 weeks.
   ii. Recurrent meningitis.
   iii. Aerocoele formed, which causes symptoms.
   iv. Constant increase in the quantity of CSF rhinorrhoea.

ORO-ANTRAL FISTULA

It is an abnormal communication between the maxillary sinus and the oral cavity.

Etiology:

1. ***Dental extraction*** – when a fistula forms through the tooth socket.
2. ***Caldwell-Luc operation*** – when the incision site and the site of opening of the maxillary antrum, overlap each other.
3. ***Erosion*** of the antrum by malignant tumours.
4. ***Penetrating wounds*** of the upper jaw.
5. ***Osteitis*** of the maxilla.
6. ***Developmental cysts.***
7. ***Radiation therapy.***

General conditions like diabetes, tuberculosis etc. act as predisposing factors.

Clinical features

1. Discharge in the mouth
2. Regurgitation of food particles and air into the nose.
3. Symptoms of maxillary sinusitis.
4. Prolapse of the antral lining may occur.
5. A probe can be passed into the antrum.

Investigations:

A fistulogram is performed by instillation of radio-opaque dye into the fistula and then an X-ray is removed to detect the connection of the fistula.

Management:

1. Antibiotics to control the acute infection.
2. Caldwell-Luc operation for removal of foreign bodies and then a primary closure of the fistula in done.
3. A small fistula is closed, after its edges are freshened by a burr or currette.
4. A large fistula requires either a buccal or a palatal flap for its closure.

The methods used to obliterate the fistula are by the use of

- Buccal flap
- Bipedicle flap
- Buccal fat and mucosal closure
- Bone graft

- Gold foil
- Island flap
- Tongue flap
- Palatal Rotation flap (Greater palatine artery)

The success of these procedures depend on the control of sinus infection, size of fistula, adequate sinus drainage, flap design and patient compliance like smoking, oral hygiene and avoiding hard foods.

NASAL OBSTRUCTION

Causes:

A) Unilateral

1. Sinusitis
2. Antrochoanal polyp
3. Deviated septum
4. Rhinolith
5. Unilateral choanal atresia
6. Foreign body
7. Rhinosporidiosis
8. Malignancy of the nose, paranasal sinuses or nasopharynx

B) Bilateral

1. Allergic and Vasomotor rhinitis
2. Ethmoidal polypi
3. Adenoids
4. Septal hematoma and abscess
5. Sinusitis
6. Atrophic rhinitis
7. Nasopharyngeal angiofibroma
8. Diphtheria
9. Rhinitis medicamentosa

NASAL DISCHARGE

Types:

1. *Mucopurulent* – infections and neoplasms.
2. *Watery* – allergic and vasomotor rhinitis, CSF rhinorrhoea.
3. *Blood stained* – foreign body, malignancy, diptheria and rhinosporidiosis.
4. *Foul smelling* – atrophic rhinitis, malignancy, sinusitis and old foreign bodies. Common causes of unilateral and bilateral nasal discharge are similar to those causing nasal obstruction.

Causes of purulent nasal discharge

ANOSMIA

Anosmia is loss of sense of smell.

Causes:

I. Intranasal airway obstruction

1. Allergy
2. Nasal polyps
3. Inflammatory exudate
4. Trauma and Synechiae
5. Malignancy

II. Intranasal mucosal destruction

1. Atrophic rhinits
2. Senile atrophy
3. Toxic chemical poisons
   i. Zinc suphate
   ii. Lead

III. Intranasal surgery which produces Olfactory nerve injury

IV. Intranasal lesions

1. Tumours, involving frontal lobes and cribriform plate.
2. Vascular lesions, causing ischemia of the Olfactory apparatus.
3. Head injury
   i. Cribriform plate fracture
   ii. Hemorrhage
   iii. Laceration of olfactory nerves.
4. Infections

V. Hysteria and malingering

CHOANAL ATRESIA

It is a congenital disorder where there is failure of canalisation of the posterior nares.

Types:

A) I. Unilateral
II. Bilateral

B) I. Complete
II. Incomplete

C) I. Bony
II. Membranous.

Etiology:

Development in origin due to the persistence of the bucco-nasal membrane.

Clinical features

1. Breathing difficulty especially when it is bilateral.
2. Difficulty in suckling leading to cyanosis during feeds
3. Unilateral atresia detected late

Investigations

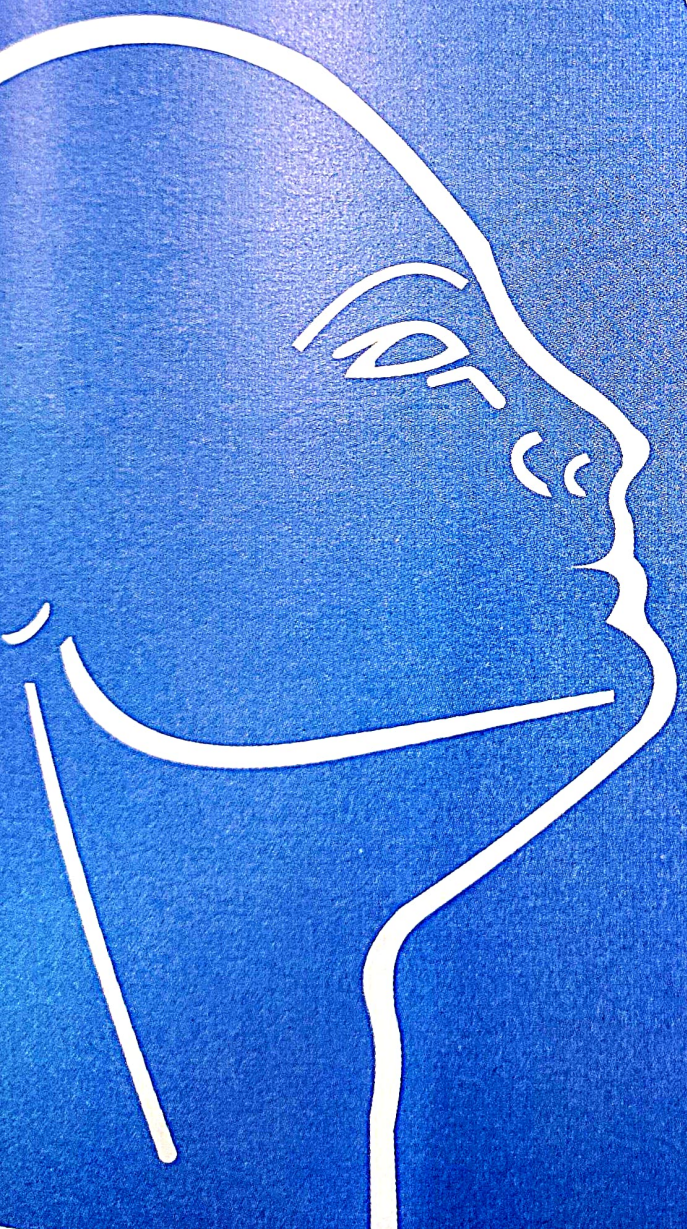
1. Soft rubber is passed along the floor of the nose, cannot enter the pharynx
2. Radio-opaque dye is instilled into the cavities and the X-ray would detect site of atresia.
3. CT scan detects whether a membranous or bony atersia is present.
4. Nasal endoscopy to detect the extent of atresia.

Management

1. Endotracheal intubation or tracheostomy may be required during the asphyxia as an emergency procedure.
2. Perforators are used to open the atretic plate.
3. Endoscopic LASER beam is used to cut the plate.

SECTION - III

ORAL CAVITY



CHAPTER 34

Anatomy of the Oral Cavity

The oral cavity starts from lips and ends at the anterior tonsillar pillar (Palatoglossus muscle).

Lips : Each lip is made up of Skin, Superficial fascia, Orbicularis oris muscle, submucosa containing labial mucous glands with blood vessels and mucous membrane.

Lips are externally lined by skin where as internally by mucous membrane.

The lips laterally meet at angles of mouth (commissure). (Situated by the side of adjacent canine teeth). The inner frenulum is attached to gums. The philtrum is the median vertical groove of outer surface. (The closed lips are referred as competent).

The central part is drained by the submental nodes and remaining part by the submandibular nodes.

The upper lip is drained by the pre-auricular, infraparotid and submandibular nodes.

1. ***Roof*** – is formed by the hard palate, which is part of the maxilla and also constitutes floor of the nasal cavity. Palatal rugae are irregular folds of mucosa .
2. ***Posteriorly*** – is the curtain like soft palate, which forms the floor of the nasopharynx. It is yellow red in colour.
3. ***Floor*** – consists of a muscular diaphragm slung between the mandible and the hyoid bone. The frenulum divides it into two parts. Sublingual glands opens on either side of frenulum. Sublingual folds covers the sublingual glands on both side of the duct.
4. ***Laterally*** – is the cheek covered by mucosa.

The cheek starts from angle of mouth to the ascending ramus of mandible. The mucosa contains yellow ectopic sebaceous glands (Fordyces granules).

TONGUE

It is a strong, muscular and mobile structure. The margin between the anterior two-thirds and the posterior one-third is delineated by large circumvallate papillae. The function of the tongue is associated with speech and the first stage of swallowing. There are four taste sensations – sweetness on the tip of the tongue, bitter at the root, salt and sour along the edges. Common sensations for the anterior two-thirds are via the lingual nerve and special senses via chorda tympani, the posterior third is supplied with common sensation via glossopharyngeal nerve (IX). Motor supply of the tongue is by hypoglossal nerve (XII).

Dentition – in the adult comprises of two incisors, one canine, two pre-molars and three molars on each side.

Salivary glands :

Gland	Duct	Opening
1. Parotid	Stenson's	Opposite upper second molor
2. Submandibular	Wharton's	Either side of frenulum
3. Sublingual	Bartholin's	Floor of mouth

Retromolar Trigone – This mucosa covered triangular area, containing pterygomandibular raphe is present on the ramus of mandible. Its apex is adjacent to the maxillary tuberocity and base is posterior to the lower alveolus. The inferior alveolar nerve lies lateral to the raphe and is the site of rapidly spreading silent malignancies.

CHAPTER 35

Diseases of Oral Cavity

STOMATITIS

It is an inflammatory condition of the oral cavity produced by local or systemic diseases.

Causes

A. Local:

1. Traumatic injuries – from jagged teeth, cheek biting, tooth brush injuries, dentures etc.
2. Thermal injuries – from hot foods.
3. Tobacco smoking.
4. Radiotherapy.

B. General:

1. Avitaminosis
   a. vitamin B-complex deficiency
   b. vitamin C deficiency
2. Infective
   a. acute ulcerative stomatitis (Vincent's angina)
   b. acute coccal stomatitis
   c. tuberculosis of the oral cavity
   d. syphilis
   e. monilia (thrush) due to candida albicans
   f. herpes simplex
   g. herpes zoster
   h. diphtheria
   i. AIDS
3. Allergic
   a. lipstick
   b. acrylic resins in dentures.
   c. aspirin and iodides
4. Metals
   a. mercury
   b. bismuth
   c. gold
   d. lead
5. Drugs
   a. antidepressants
   b. eptoin.

Trismus

It is the inability to open the mouth. It may be caused by generalised spasm of the muscles of mastication, preventing opening of the mouth.

Etiology:

A. General causes:

1) Tetanus
2) Strychnine poisoning
3) Tetany
4) Hysteria
5) Scleroderma

B. Local causes:

1) Quinsy
2) Alveolar infection
3) Acute parotitis
4) Arthritis of temporomandibular joint
5) Acute otitis externa
6) Mikulicz's syndrome
7) Mumps
8) Submucous fibrosis
9) Ankylosis of temporomandibular joint
10) Malignancy of cheek, tonsil, palate, maxillary sinus
11) Burns

Complications:

1) Oral hygiene is affected.
2) Nutrition may suffer
3) Examination of oral cavity, pharynx and larynx becomes difficult.
4) Endotracheal intubation is a problem in such patients.

Management:

1) Reassurance
2) Feeding from side of the mouth
3) Nasogastric feeds
4) Muscle relaxants
5) Surgical – Temporomandibular joint replacement.
6) LASER – division of pterygo mandibular raphe.

APHTHOUS ULCERS

Recurrent aphthous ulcers is the commonest disease which affects the oral mucosa.

Etiology

1. Nutritional deficiency
2. Hormonal
3. Psychogenic
4. Infections
5. Auto-immune reaction.

Clinical features

Occurs anywhere inside the lips or buccal mucosa, floor of mouth or edge of tongue. They are quite sensitive and painful, have a grey base with a surrounding red circumference and disappear after a few days with or without treatment. Large ulcers may cause scarring after the healing process.

Management:

1. Antiseptic mouth washes.
2. Topical steroids
3. Vitamins
4. Antibiotics to combat the anaerobic infection – Metronidazole.
5. Silver nitrate cauterisation of edges.

LEUCOPLAKIA

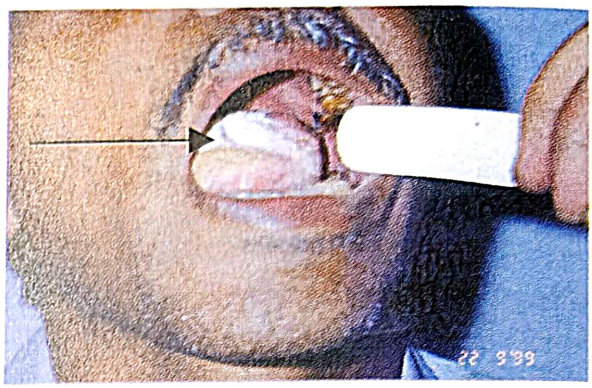
Leucoplakia is a white patch on the mucosa of the mouth or tongue.

Predisposing factor

1. Smoking
2. Sepsis
3. Sharp tooth
4. Spices
5. Spirit
6. Syphilis
7. Betel nut
8. Poor oral hygiene
9. Chronic infection.

Clinical features

Patient is usually asymptomatic but its importance lies in the fact that a malignant change may occur. Biopsy must be taken on clinical judgement to rule out malignancy.



Stages of Leucoplakia leading to Malignancy:

1. ***Hyperkeratosis :*** Mild thickening of the surface producing a milky film. There is a more rapid multiplication of epidermal cells than the normal.
2. ***Acanthosis :*** Chronic inflammatory changes are seen in the dermis and there is a proliferation of the prickle cell layer.
3. ***Parakeratosis :*** Affected areas appear as if covered with smooth paint and the cells are not mature even when they reach the superficial layer.
4. ***Widening of Rete Pegs :*** The surface becomes irregular resembling wrinkled paint and there is a widening of the downward projections (rete pegs) of the epidermis.
5. ***Dyskeratosis :*** The affected area assumes a warty appearance and the disorganisation of the epidermis eventually becomes greater.
6. ***Cancer in situ :*** Epithelial pearls are seen on the epidermis and the appearance of this stage shows warty projections. Here there are no signs of infiltrations and it is also known as intra-epidermal cancer.

Treatment

1. Avoidance of predisposing factors and regular follow up.
2. Excision of leucoplakic patch following a positive malignancy report.

MALIGNANCY OF THE TONGUE

The incidence of Carcinoma of the Tongue is particularly common due to the habits of paan and tobacco chewing.

Etiology

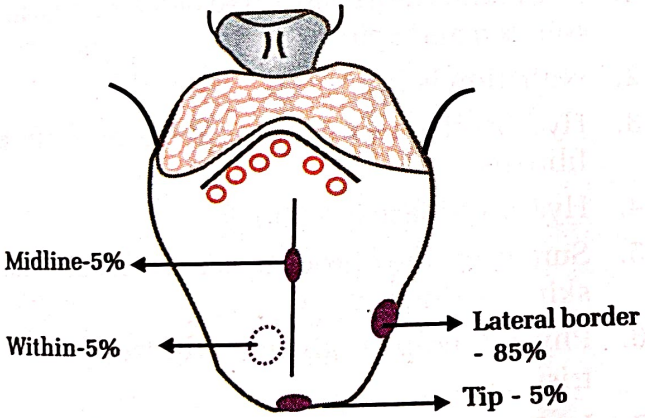
1. Age – Seen usually after the age of 35 years.
2. Sex – No particular predeliction
3. Precipitating factors –
   - Smoking
   - Sepsis
   - Spirits
   - Tobacco
   - Spices
   - Sharp tooth
   - Syphilis
   - Avitaminosis
   - Plummer – Vinson Syndrome

Gross Pathology

a. Ulcer
b. Warty growth
c. Infiltrative mass

Pre-malignant conditions

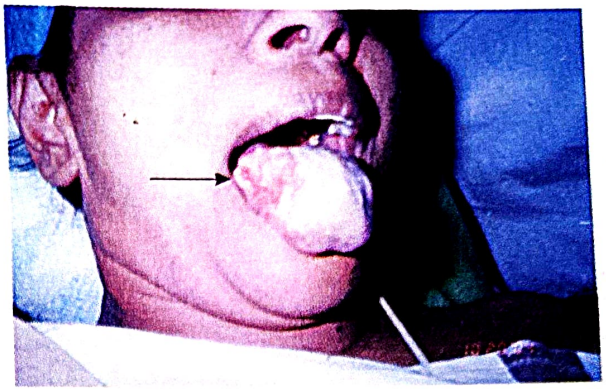
a. Leucoplakia
b. Erythroplakia
c. Melanoplakia
d. Submucous Fibrosis



Sites of malignancy

Clinical features:

1. *Pain* – usually referred to the ear
2. *Fetor* – foul odour.
3. *Excessive salivation*
4. *Odynophagia* – pain during swallowing
5. *Speech difficulties*
6. *Ankyloglossia* – restricted tongue movement.
7. *Neck swelling* – due to lymph node metastasis.



Malignancy of (R) Lateral border of Tongue

Spread

1. ***Local*** : Spread occurs easily as the interlacing muscle fibres of the tongue form an easy pathway for the spread of cancer and the constant movement of the tongue also disseminates the cancer widely.
2. ***Lymphatic*** : Spread to submental, submandibular and the upper cervical group of lymph nodes.
3. ***Hematogenous*** : To bone (mandible).

Staging:

TNM Classification

Tumour :

T_0 - no evidence of primary tumour
T_1 - tumour is 2 cm or less in its greatest diameter
T_2 - tumour is greater than 2 cm but not more than 4 cm in its greatest diameter
T_3 - tumour is more than 4 cm in its greatest diameter.
T_4 - involvement of adjacent areas.

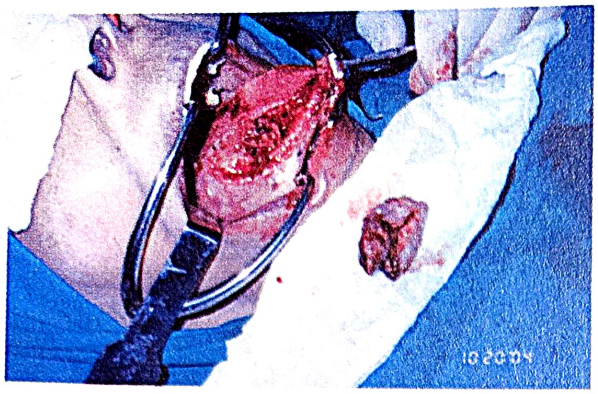
Exact size of tongue tumours is obtained only by palpation.

Node and Metastasis – Same as for carcinoma of the Larynx.

Treatment:

Stage

I - Surgery (Local excision)
II - Surgery (Hemi-Glossectomy with neck dissection)
III - Radiotherapy (Brachytherapy)
IV - Combined modality treatment



Local excision of lateral border of tongue using LASER

Cause of Death

a. Aspiration
b. Hemorrhage
c. Cachexia
d. Metastasis.

ORAL SUBMUCOUS FIBROSIS

This is a chronic disease of unknown etiology, characterised by fibrosis in the submucosal layers of the oral cavity affecting mainly the soft palate, cheek and tonsils.

Etiology

1. Age – Seen around the age of 18 to 40 years.
2. Sex – females are more commonly affected.
3. Widespread in India and Pakistan.
4. Predisposing factors –
   i. Local irritation by betel nuts, paan, tobacco, chillies and spicy food.
   ii. Trauma due to sharp edges of betel nuts causing multiple puncture sites, ulceration and healing by fibrosis.
   iii. Vitamin deficiency.
   iv. Local infections.
5. Localised collagen disorders.

Pathology

Stages:

1. Stomatitis and vesication
2. Fibrosis
3. Malignancy.

Clinical features

1. Soreness of mouth with constant burning sensation.
2. Trismus occurs due to involvement of retromolar trigone area or the pterygo-mandibular raphe.
3. Vesicle formation.
4. White discoloration.
5. Fibrosis may spread to soft palate & restrict tongue mobility.

Differential diagnosis

1. Trismus – its causes
2. Leucoplakia
3. Thrush
4. Lichen planus.

Investigations

1. Complete blood count
2. Biopsy
3. Gastric analysis

Treatment

1. Avoidance of irritants like betel nuts, paan, tobacco and spices.
2. Nutrition is improved.
3. Hydrocortisone injections to prevent further fibrosis.
4. Hyaluronidase injections.
5. Surgically, the fibrous bands are excised and skin grafting is done.
6. Physiotherapy is given for the treatment of trismus.
7. LASER is used to cut the fibrous bands.
8. Condylar surgery - Temporal graft

Prognosis

Submucous fibrosis is a pre malignant condition, therefore a regular follow up is necessary and the causative agents are to be avoided.

LUDWIG'S ANGINA

It is the infection of the submandibular space, which lies between the mucous membrane of the floor of the mouth and tongue on one side,

and superficial layer of deep cervical fascia extending between the hyoid bone and mandible on the other.

This space is subdivided by the mylohyoid muscle into sublingual (above it) and submaxillary (below it). These two compartments are continuous around the posterior border of the muscle.

Etiology

1. Dental infections : roots of premolars cause sublingual space infection while roots of molar cause submaxillary space infection.
2. Submandibular sialadenitis
3. Injuries of oral mucosa
4. Fracture of the mandible

Clinical features:

1. Swelling which is tender.
2. Odynophagia (difficulty in swallowing) with varying degree of trismus.
3. Breathlessness.
4. Fever

Complications:

1. Spread of infection to parapharyngeal and retropharyngeal spaces and thence to the mediastinum.
2. Airway obstruction due to laryngeal oedema, or swelling and pushing back of the tongue.
3. Septicaemia
4. Aspiration pneumonia.

Management :

1. Systemic antibiotics
2. Incision and drainage of abscess : Intraoral incision is taken if infection localised to sublingual space or if submaxillary space involved then external transverse incision from one angle of mandible to the other is made with vertical opening of musculature of the tongue. Always to be performed under local anaesthesia.
3. Tracheostomy if airway obstruction.

CHAPTER 36

Disorders of Salivary Glands

Mumps

It is a non-suppurative acute systemic communicable viral infection of childhood. (Paramyxo - virus).

The incubation period is about 14 – 18 days. One attack of mumps gives life-long immunity. It spreads by droplet nuclei, saliva and fomites. It mainly affect the meninges, pancreas and gonads. It does not produce pus & occurs in epidemics (which help to differentiate it from bacterial parotitis)

Clinical features – (Commonly both sides involved)

- Fever, Malaise, Headache, Anorexia.
- Painful enlarged parotid glands.
- Earache.
- Difficulty to eat.

Signs :

- Swelling of parotid gland which is tender.
- Obliteration of space between the ear lobe and the angle of mandible.
- The parotid duct (Stenson's duct) opening is red congested and edematous.

Diagnosed by elevated level of serum amylase and isolation of virus from urine 6 days before and upto 13days after the appearance of symptoms. Cerebrospinal lymphocytosis seen in these (65%) patients.

Prevention :

Prevented by giving live attennuated mumps vaccine (Jeryl Lynn Strain) subcutaneously at the age of 1yr in children. It is given in the national program during 12 to 15 months subcutaneously. (MMR vaccine).

Management :

1) Good oral hygiene
2) Antipyreties (Paracetomol but avoid aspirin)
3) Warm or cold compresses of parotid gland

PAROTITIS

Acute suppurative parotitis :

Acute Bacterial infection of parotid gland occurs in chronic debilitated, immunodeficient, anorexic patients. It is usually limited to only one gland.

Etiology :

1. Secondary to obstruction of the stensen's duct which may be due to decreased salivary flow (allows the ascent of a bacterial flow) or by parotid calcular and epithelial debris (remnants) or by a duct stenosis due to dental problems.
2. The causative organism are staphylococcus aureus, Streptococci, E. coli etc.
3. The chronic parotitis is most often due to Tuberculosis, Actinomycosis, Leprosy, Tularemia or Sarcoidosis.
4. Actinomycosis may present as on parotid abscess with granules discharging.

Clinical Features :

- High degree fever
- Unilateral tender enlargement of parotid gland
- Pain is severe and aggravated by Jaw movement
- Trismus (spasm of masseter Temporalis and pterygoid muscles)

Signs :

The parotid duct opening is edematous and red, on pressure pus can be seen discharging from the duct opening.

Investigation :

1. Elevation of white cell count. Increase in neutrophils.
2. Culture the pus and identify the causative organism and treat it.
3. Ultrasonography will help to identity the site of pus collection, which can be aspirated.

Treatment :

1. Adequate hydration
2. Oral hygiene
3. Parental antibiotic given based on the culture sensitivity report
   - Cephalosporins
   - Vancomycin hydrochloride
   - Clindamycin hydrochloride
   - Cefoxin sodium
   - Metronidazole + Imipenem
   - Penicillin + B lactamase inhibitor
4. Surgical drainage

Parotid Abscess

The unresolved parotitis may form into parotid abscess. Due to dense fibrous capsule, pus cannot come in the mouth in early stage. This abscess may spread into Temporomandibular joint, into ear or face, or may cause septic arthritis or osteomyelitis of mandible or into the chest. It can be diagnosed by ultrasonography.

The treatment is incision and drainage under anesthesia. A vertical preauricular incision is taken. Then abscess is opened horizontally in the direction of branches of facial nerve to prevent their injury.

Parenteral Antibiotics given to control infection, based on culture sensitivity.

SIALOLITHIASIS

The formation of stone in the salivary gland is known as sialolithiasis. The gland involved are submandibular (78%) and parotid glands. There are solitary and unilateral.

The submandibular gland is a mixed seromucinous gland, containing high calcium. The epithelial debris calcifies easily and has high density, so they are radio-opaque.

The parotid gland is a serous gland and is low in calcium. Here the epithelial debris remains as sludge and blocks the ductal opening. So they soften and do not impact and come out so easily. These also have lower density and are radio-lucent.

Symptoms:

1. Asymptomatic
2. Pain & swelling of involved gland - the pain appears before the start of meals (stimulation of saliva discharge and gland swelling due to obstructed duct)
3. Citrous drinks or fruits cause more pain, as they increase the salivary stimulation.
4. The secondary bacterial infections will lead to abscess formation.

Signs :

Sometimes stone can be seen in the duct opening or can be palpated in the duct area. There will be swelling of gland with pain. (Which is aggravated by eating).

Localized tenderness in the floor of mouth.

Stone can be palpated bimanually.

The stone in the anterior 2/3rd of submandibular gland can be seen by true occlusal radiographs and the distal oblique occlusal radiographs are used for examining stone in the posterior third of the duct. It is always better to perform both views, to rule out multiple stones.

When no stones are seen on the X-rays one should suspect the possibility of a radiolucent

stone in the gland and sialogram should be done to detect radiolucent stone.

Treatment:

The stones in the duct opening can be removed by sialodochotomy (trans-oral removal of stone).

The stones found in the gland can be treated by excising the gland.

SIALECTASIS

Sialectasis is a cystic dilatation of salivary glands, formed by progressive necrosis and disintegration of the tissues. The debris blocks the duct of the gland and causes hypertrophy (enlargement of gland), ductal dilatation and stenosis.

The pathogenesis is similiar to bronchiectasis and is diagnosed by sialogram which shows cystic sialectasis (i.e. large cysts with stenosed, dilated duct).

SJOGREN'S SYNDROME

It is an autoimmune disorder of exocrine glands. The patients may have other connective tissue diseases like Rheumatoid arthritis, SLE, Polymyositis, Polyarteritis nodosa.

The parotids are affected more commonly.

Clinical features - Dry mouth, Painful tongue, Itching gritty and dry Eyes. (Stomatitis Glosssitis).

The diagnosis is made by lower lip mucosal biopsy which shows lymphocytic infiltration.

SALIVARY FISTULAE

These arise from parotid and submandibular gland. These fistulae may drain into the mouth or externally outside the skin.

Etiology:

1. Post surgery
2. Injury to face
3. Sepsis of salivary gland

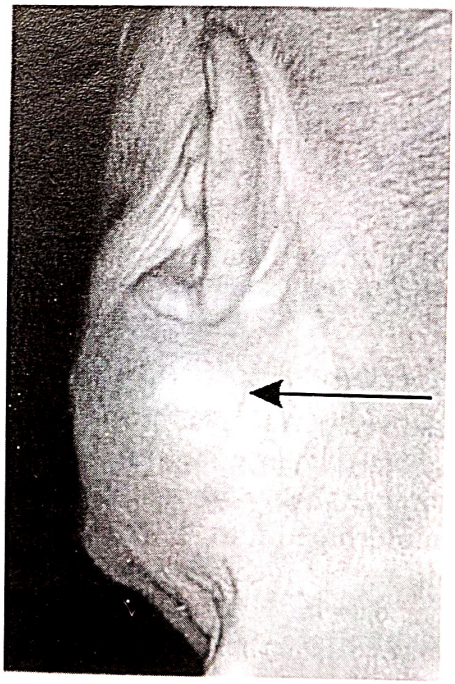
Symptoms :

- Non healing sutures on the gland.
- Leaking of secretions / saliva.
- Leaking of saliva from parotid by food thought (dry facial wound).
- Small fistulas respond to pressure dressing.

These are treated by excision of gland. Correction of damaged duct, ductal ligation of drainage duct opening into the mouth. If not responding by above methods one should consider total excision of the gland.

SALIVARY GLAND TUMOURS

Salivary gland tumours most commonly affect the parotid and less commonly submandibular gland and submucosal glands. It comprises of 3% of all tumors. About the 80% are benign and rest 20% are malignant.



Left Parotid tumor

Benign Tumors:

- Pleomorphic adenoma (Mixed Parotid tumor)
- Warthin's tumor
- Oxyphill adenoma (Oncocytoma)
- Hemangioma

Pleomorphic adenoma

These are slow growing, benign mixed epithelial tumor containing mesenchymal and epithelial cells.

Warthin's tumors (Papillary Cystadenoma)

It occurs in tail of parotids in middle aged males. These arise in the salivary duct which are trapped in the intraparotid lymph nodes. (Lower pole of parotid).

Treated by doing a wide surgical excision.

Oncocystoma

These originate from external (outer) part of parotid gland, composed of pleomorphic eosinophilic cells.

These benign tumours are slow growing, fusiform, firm to hard, nodular tumors. They are ballottable on bimanual palpation.

Malignant Tumors:

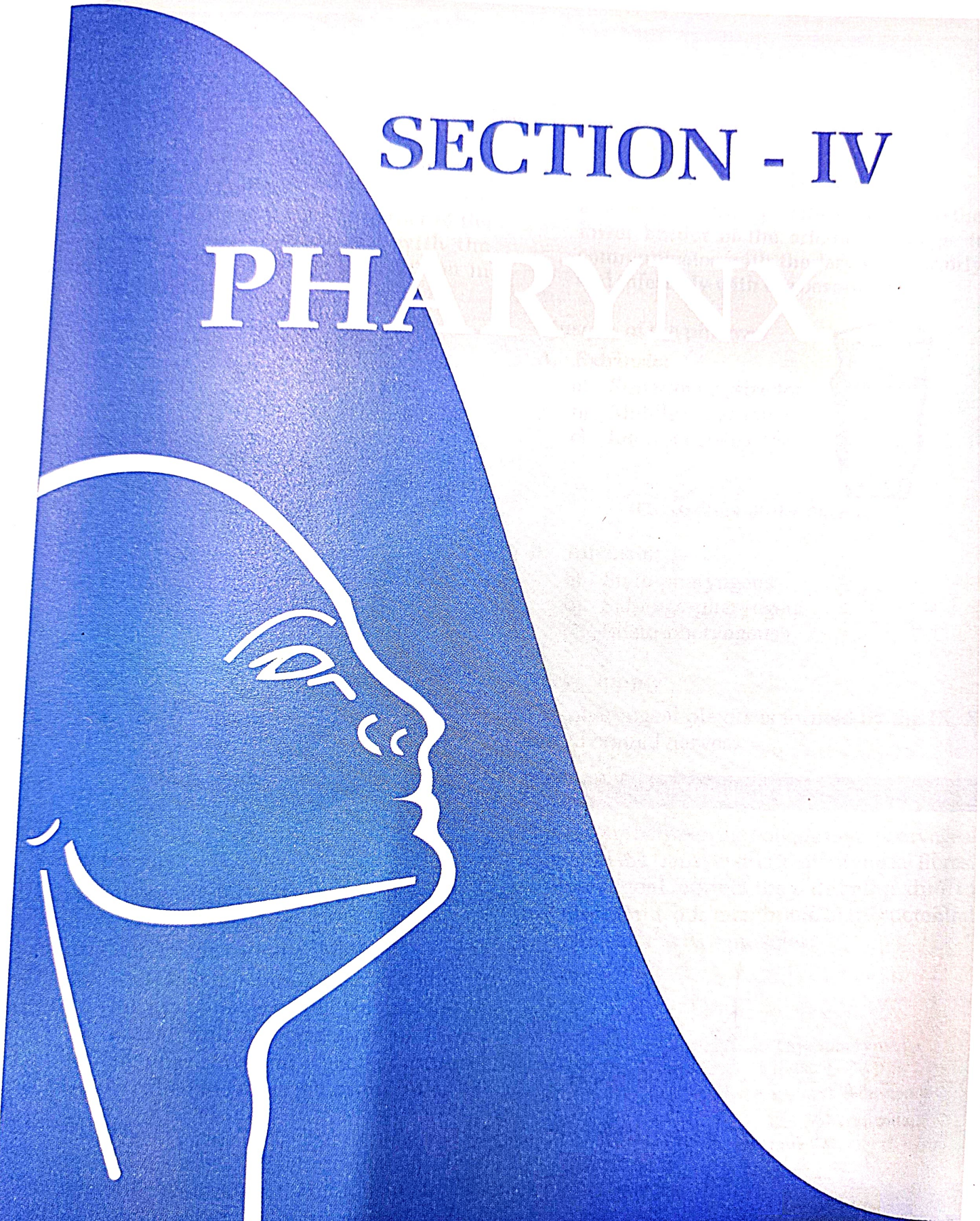
- Mucoepidermoid
- Acinic cell
- Adenoid cystic
- Adenocarcinoma
- Lymphoma

Malignant salivary gland tumours are found mainly in parotid followed by submandibular gland.

Treatment consists of a Total Parotidectomy with sacrificing the facial nerve and post operative radiotherapy may be necessary.

SECTION - IV

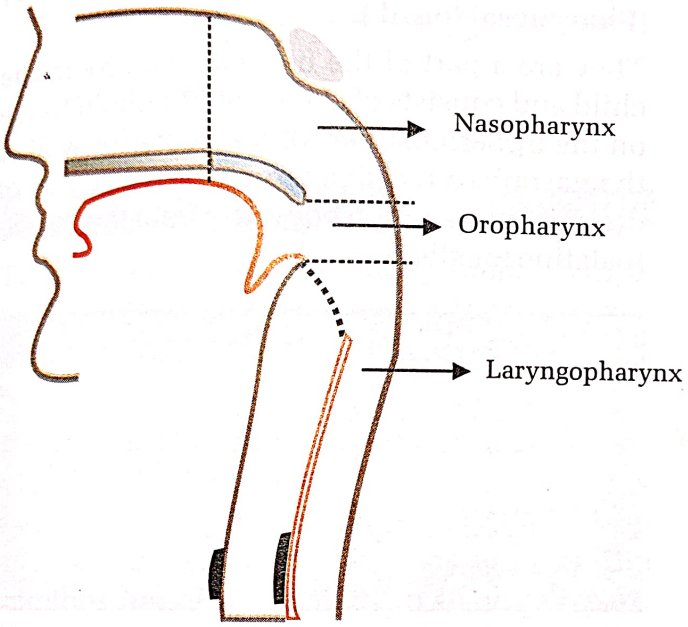
PHARYNX



CHAPTER 37

Anatomy of the Pharynx

The pharynx is a fibromuscular tube extending from the base of skull above to the 6th cervical vertebra and below at the lower border of the cricoid where it is continuous with the oesophagus. The pharynx is about 10 cm in length in the adult.



Parts of pharynx

Parts:

1. ***Nasopharynx***

   At the junction of the roof and the posterior wall of the nasopharynx is an aggregation of lymphoid tissue called adenoids. In the anterior wall open the turbinates and below them is the attachment of the soft palate. On the lateral wall are openings of the eustachian tubes and behind each of these is a hollow called the fossa of Rosenmuller.

2. ***Oropharynx***

   Extends from the soft palate to the tip of the epiglottis. Anteriorly it communicates with the oral cavity, while inferiorly with the hypopharynx.

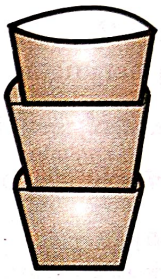
3. ***Hypopharynx or Laryngopharynx***

   Extends from the tip of the epiglottis to the lower border of the cricoid cartilage. It communicates with the larynx anteriorly and inferiorly with the oesophagus.

Muscles of the pharynx

A. Extrinsic:

a) Superior constrictor
b) Middle constrictor
c) Inferior constrictor



Constrictors of the Pharynx

B. Intrinsic:

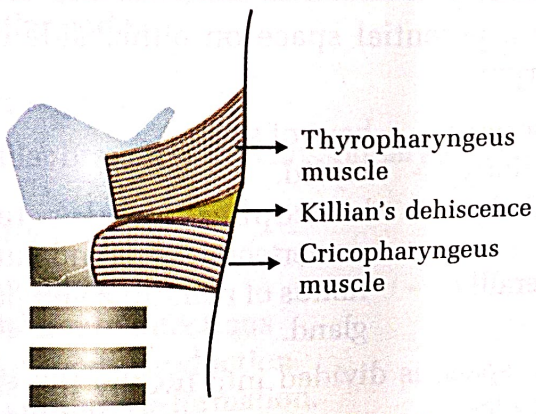
a) Stylo-pharyngeus
b) Salpingo-pharyngeus
c) Palato-pharyngeus

Nerve Supply

The pharyngeal plexus is formed by the IX, X and XI cranial nerves.

KILLIAN'S DEHISCENCE

This is a gap between the oblique thyropharyngeal fibres and the transverse cricopharyngeal fibres. A pharyngeal pouch may develop due to prolapse of mucous membrane in this potential gap between the two muscles.



Killians dehiscence (shaded)

RETROPHARYNGEAL SPACE

This is a potential space between the posterior pharyngeal wall and the prevertebral muscles. The space is divided into two compartments by a median raphe, (**space of Gillette**).

Anteriorly – formed by the buccopharyngeal fascia covering the posterior pharyngeal wall.

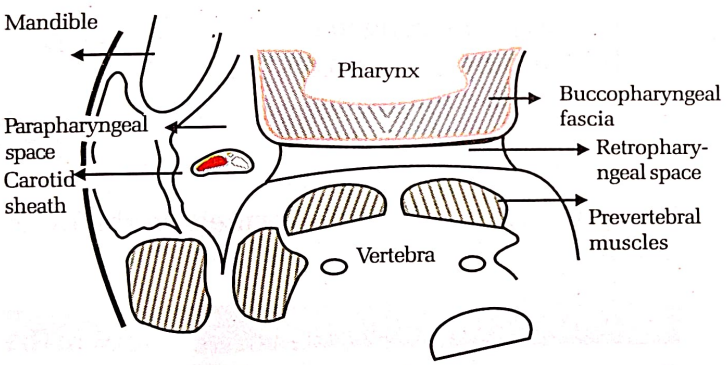
Posteriorly – formed by the prevertebral fascia covering the cervical vertebra and the prevertebral muscles.

Laterally – is the carotid shealth and the parapharyngeal space.

Superiorly – is the basiocciput.

Inferiorly – superior mediastinum.

Contents – the retropharyngeal lymph nodes which disappear by the age of 4 years.



Neck spaces

PARAPHARYNGEAL SPACE

It is a potential space on either side of the pharynx.

Superiorly - base of skull.
Inferiorly - Hyoid.
Medially - buccopharyngeal fascia, prevertebral fascia and muscles.
Laterallly - ramus of mandible and parotid gland.

The space is divided into two by the styloid muscles.

Contents:

i. Great vessels of the neck in the carotid sheath.
ii. Deep cervical lymph nodes.
iii. IX, X, XI and XII cranial nerves.
iv. Cervical sympathetic trunk.

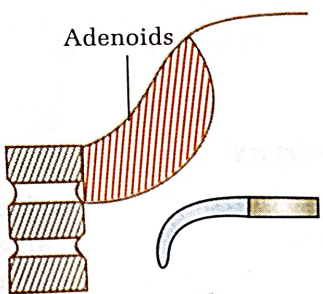
Sinus of Morgagni

It is the space in between superior constrictor muscle and base of skull, through this eustachian tube enters the nasopharynx.

ADENOIDS (Nasopharyngeal tonsil) (Pharyngeal tonsil)

They are a part of the normal anatomy in the child and consists of a mass of lymphoid tissue on the uppermost part of the posterior wall of the nasopharynx. They form the inner part of the Waldeyer's ring. They differ from the Tonsils (palatine tonsils).

Adenoids	Tonsils
1. Single	Paired
2. No Capsule	Capsule
3. Ciliated columnar	Stratified squamous epithelium
4. No crypts	Crypts
5. Involution occurs by puberty	Persist



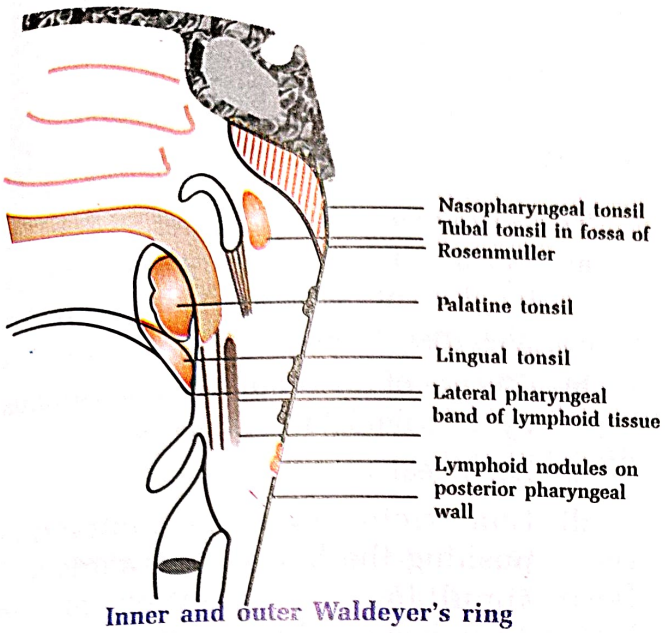
Adenoids

Adeoids are supplied by the Ascending pharyngeal artery (ECA) and drained by the pharyngeal plexus of veins.

Lymphatic drainage - These are drained into node of Rouviere (Lateral retropharyngeal nodes) (Efferents) No afferents.

Site – Fossa of Rosenmuller

– Deep Cervical group of lymph nodes
– Posterior triangle of neck .



Inner and outer Waldeyer's ring

TONSILS (PALATINE TONSILS)

The palatine tonsil is a subepithelial lymph node with prominent germinal centres concerned with the production of lymphocytes and plasma cells.

The tonsil is of the size of an almond and its medial surface is lined by stratified squamous epithelium and about 15-30 crypts extend deep into its tissues. The crypta magna or the intratonsillar cleft is found in its upper part. The peritonsillar space separates the tonsillar capsule from the superior constrictor muscle.

Relations –

Anteriorly – Palatoglossus muscle (Anterior pillar)

Posteriorly – Palatopharyngeus muscle.

Superiorly – Soft palate

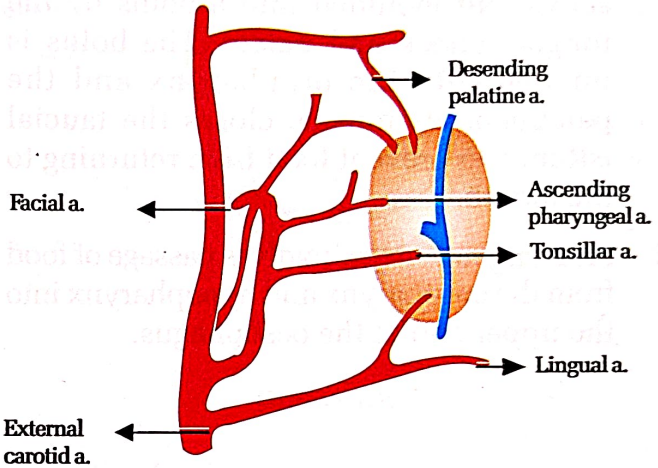
Inferiorly – Posterior 1/3rd of tongue.

Laterally – (Bed of tonsil) From midline to outside :

- Loose areolar tissue and Para tonsillar vein
- Pharyngeal aponeurosis. Superior constrictor.
- Buccopharyngeal fascia.
- Glossopharyngeal neve.
- Pterygoid muscles
- Mandible

Blood supply

1. The tonsillar branch of the facial artery branch of ECA
2. Ascending pharyngeal artery – External carotid artery
3. Ascending palatine artery – branch of facial artery (Facial artery – ECA)
4. Dorsal lingual branches of the Lingual artery (ECA)
5. Descending palatine artery – (Maxillary artery – ECA)



Blood supply of tonsil

Venous drainage

Into the paratonsillar vein which drains to the pharyngeal plexus and ultimately to the internal jugular vein.

Lymphatic drainage

Jugulodigastric lymph node also called the tonsillar node.

Nerve supply

Tonsillar branch of the Glossopharyngeal nerve.

Functions:

1. Immunity
2. Barrier to infections
3. Antibody production
4. Lymphocyte formation.

CHAPTER 38
Deglutition

Deglutition is a co-ordinated reflex whereby food is carried from the oral cavity, after mastication into the stomach.

Stages :

1. ***Oral*** – part is a voluntary stage where food is broken down by chewing lubricated by saliva and moulded into a bolus by the tongue, cheek and palate. The bolus is pushed into the oropharynx and the palatoglossus muscle closes the faucial isthmus to prevent food from returning to the mouth.
2. ***Pharyngeal*** –stage involves passage of food from the oropharynx and hypopharynx into the upper end of the oesophagus.

The reflexes which follow are:

a) Closure of the nasopharyngeal isthmus by the soft palate on the posterior pharyngeal wall.

b) Closure of the oropharyngeal isthmus by the palatoglossus muscle.

c) Laryngeal inlet closure.

d) Constrictor muscles contraction pushing the bolus downwards and simultaneous relaxation of the cricopharyngeal sphincter.

3. ***Oesophageal*** – stage is involuntary and food passes down by peristalsis. Gravity plays little part in the passage of food. The bolus of flood takes 3-5 seconds to reach the stomach.

CHAPTER 39

Examination of the Throat

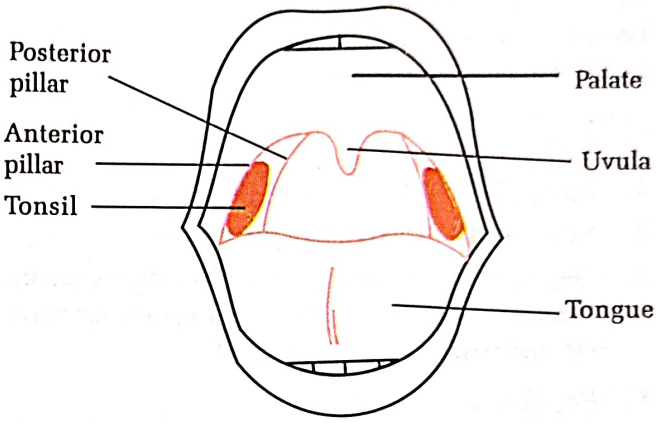
History

Complaints:

Throat pain	- Localised / generalised / referred, continuous / intermittent, dull aching / sharp shooting, effect with analgesics
Hoarseness of	- Sudden / gradual, continuous / intermittent diurnal variation, vocal overuse / vocal abuse, voice with colds
Fever	- Associated with throat pain, high grade / low grade, continuous / intermittent
Difficulty in swallowing	- Sudden / gradual, solids / liquids, progressive / same, associated with vomiting.
Difficulty in breathing	- On exertion, noisy breathing
Neck swellings	- Localised / generalised, with fever / throat pain, sudden / gradual, size is constant / decreased / increased

H/O Treatment taken

H/O Vocal abuse / trauma / operations, symptoms suggestive of Tuberculosis, Tobacco / paan / alcohol addiction.



Oral cavity

EXAMINATION

1. Oral cavity – for lips, teeth , tongue, cheek, floor of mouth and openings of salivary glands.
2. Oropharynx – for anterior and posterior pillars, tonsils, uvula, posterior pharyngeal wall and the gag reflex is tested.
3. Hypopharynx – by indirect laryngoscopy.
4. Neck – palpation to check for lymphadenopathy and malignancy.

CHAPTER 40

Tonsillitis

ACUTE TONSILLITIS

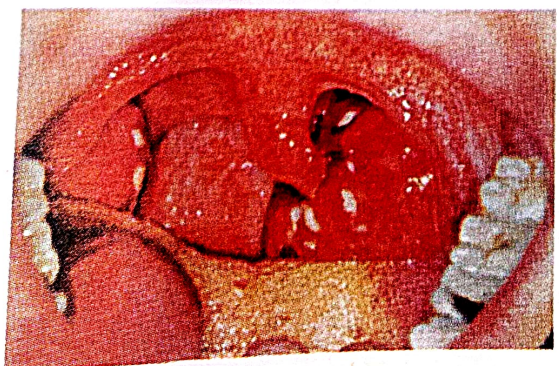
Acute tonsillitis is a disease of sudden onset with a clinical triad of sore throat, fever and malaise and even if untreated runs a relatively benign course of a week to ten days. Response to antibiotics is usually rapid and dramatic.

Etiology

1. *Age* – commoner in children
2. *Sex* – both are equally affected
3. *Organisms* – Streptococcus, Staphylococcus, Pneumococcus etc. Viruses like adenovirus, rhinovirus and enterovirus.
4. *Predisposing factors*
   i. Ingestion of cold foods.
   ii. Contact with infected persons.
   iii. Polluted ill-ventilated environment.
   iv. Pre-existing upper respiratory tract infection.
   v. Sinusitis.
   v. Lowered general resistance.

Types:

1. *Acute Follicular tonsillitis* – where inflammatory exudate from the crypts marks the reddened surface with whitish spots.
2. *Acute Parenchymatous tonsillitis* – when the whole tonsil is uniformly congested.
3. *Acute membranous tonsillitis* – in which exudate from the crypts coalesces to form a membrane over the surface.



Follicular Tonsillitis

Symptoms:

i. Throat pain – aggravated on swallowing.
ii. Fever – may be high grade.
iii. Malaise and headache.
iv. Neck swelling - jugulodigastric gland involvement.
vi. Earache – referred via glossopharyngeal nerve.
vii. Difficulty in swallowing.

Signs:

i. Tachycardia and fever
ii. Tonsils are enlarged and congested
iii. Pharynx is often inflammed
iv. Tender and enlarged jugulo-digastric lymph nodes.
v. Breath is foetid and tongue is coated.

Complications of Acute Tonsillitis

I. Local:

1. *Chronic Tonsillitis*

   The Tonsils do not always return to a state of normalcy after repeated attacks of acute tonsillitis. Minute abscess may form in the lymphoid follicles surrounding the crypts and possibly become walled-off by fibrous tissue.

2. *Quinsy (Peritonsillar abscess)*

   There is spread of infection from the tonsil with pus formation into the areolar space between the capsule of the tonsil and the tonsillar bed.

3. *Parapharyngeal abscess*

   The infection may spread from the tonsil through the superior constrictor muscle (tonsil bed) and result in pus accumulation between this muscle and the deep cervical fascia.

4. ***Suppurative cervical lymphadenitis***

   After attack of acute tonsillitis , suppuration may occur in the lymph nodes draining the area (jugulo-digastric). With the advent of antibiotics this complication is now rarely seen.

5. ***Acute otitis media***

   Spread of infection from an acute tonsillitis leading to nasopharynx would lead to infection traversing along the eustachian tube into the middle-ear.

II. **Systemic**

1. Rheumatic fever
2. Acute glomerulonephritis
3. Sydenham's chorea
4. Infective endocarditis

Differential Diagnosis of Enlarged Tonsils:

1. ***Acute Tonsillitis*** – It is a disease of sudden onset with a clinical triad of sore throat, fever and malaise. Responding rapidly to antibiotics, however if untreated has a benign course of within a week to ten days.

2. ***Infectious mononucleosis*** – Usually seen in young adults. Fever persists despite antibiotic therapy and the enlarged tonsils are covered by a membrane. Lymph node enlargement is also seen in other parts of the body and the diagnostic test is presence of antibodies by the Paul-Bunnell test.

3. ***Diphtheria*** – Quite unlikely to be confused with acute tonsillitis. It is slower in onset, first accompanied by fewer constitutional disturbances and less of local discomfort. The membrane extends beyond the tonsil surface onto the palate, which is dirty grey in colour and on removal causes bleeding. The identification of Klebs-Loffler bacillus in the membrane is diagnostic.

4. ***Leukemia*** – Results in necrotising lesions of tonsil and may present with acute sore throat. Ulceration is seen elsewhere in the mouth and oropharynx with an absence of surrounding inflammatory reaction. The general condition of the patient here, renders them unlikely to be confused with acute tonsillits.

5. ***Vincent's Angina*** – This is subacute tonsillitis with ulceration. The sloughing membrane forms an ulcer and the patients breath is characteristically foetid. The diagnosis is made from the organism being cultured from throat swab.

6. ***Thrush*** – Seen in patients having uncontrolled diabetes or being in a severly immuno-comprised state. The fungal organism is candida albicans and white patches are seen on the tonsil.

7. ***Scarlet fever*** – It is streptococcal tonsillitis and also cause punctuate erythematous rashes. The tonsils are intensely red in colour along with which the patient has a stippled palate and a 'strawberry tongue'.

8. ***Keratosis of the Tonsil*** – Heaps of epithelium on the tonsillar surface are seen generally in debilitated patients. Patients are asymptomatic and therefore no treatment is required.

9. ***Traumatic ulcer*** – injury to the oropharynx may occur accidentally by tooth brush or finger resulting in formation of a membrane.

10. ***Aphthous ulcer*** – may involve any part of the oropharynx. Their size is variable and are very painful on touch.

11. ***Malignancy of tonsil*** – Lymphomas or squamous cell carcinomas may occur on the tonsil (unilateral) presenting as an ulcerated lesion.

	Acute Follicular Tonsillitis	Diphtheria
i. Age	Any age	Younger children
ii. Onset	acute	slow
ii. Temperature	high	low
iv. Constitutional symptoms	more	less
v. Throat pain	more	less
vi. Toxemia	minimal	in late cases
vii. Tonsils	enlarged and congested	normal
viii. Membrane	- multiple follicles may coalesce	single
	- whitish colour	greyish
	- limited to tonsils	may extend to pillars and pharynx
	- thin	thick
	- easily removable	bleeds on removal
	- bilateral	may be unilateral
ix. Swab	B Hemolytic streptococci	C. Diphtheria
x. Albuminuria	absent	present
x. Lymph nodes	tender and enlarged	absent

Treatment

1. Bed rest.
2. Plenty of fluids and soft solid floods.
3. Analgesics to reduce fever and throat pain.
4. Antibiotics. A short course of a narrow spectum antibiotic in a high dosage is better than a prolonged course of a broad-spectum one in low dose.
5. Gargles.

CHRONIC TONSILLITIS

Symptoms

1. Recurrent throat pain
2. Cough
3. Neck swelling (lymph nodes).

Signs

1. Tonsils are either hypertrophied or fibrotic and small.
2. Retention cysts may be seen on the surface.
3. Cheesy material is expressed from the crypts.
4. Persistent enlarged jugulodigastric lymph nodes.

Management

1. High protein diet and improvement of general condition.
2. Antibiotics and analgesics during an acute attack.
3. Tonsillectomy.

CAUSES OF WHITE PATCHES ON TONSILS

I. Congenital

Tonsillar cyst

II. Inflammatory

i. Acute Tonsillitis
ii. Diphtheria
iii. Thrush
iv Herpes
v. Vincent's angina
vi. Tuberculosis
vii. Syphilis

III. Traumatic

i. Post-tonsillectomy slough
ii. Foreign body
iii. Thermal injuries
iv. Radiotherapy

IV. Neoplastic

i. Papilloma
ii. Polyps
iii. Lymphoepithelioma
iv. Carcinoma

V. Miscellaneous

i. Tonsillolith
ii. Tonsillar keratosis
iii. Aphthous ulcer

CHAPTER 41

Adenoids

The term adenoids is used for enlarged or hypertrophied nasopharyngeal tonsil due to repeated upper respiratory tract infections.

Etiology – Similar as for tonsillitis.

Clinical features:

I. ***Due to nasal obstruction*** – mouth breathing, nasal discharge and voice becomes nasal (rhinolalia clausa).

II. ***Due to eustachian tube blockage*** – earache, deafness, secretory otitis media and later chronic suppurative otitis media.

III. ***Due to mouth breathing*** – dribbling of saliva, noisy breathing at night, high arched palate and chronic pharyngitis.

IV. ***General*** – mental backwardness and lethargy.

Diagnosis

1. *Adenoid Facies* – develops due to chronic infection of the adenoids, i.e.
   i. Open mouth
   ii. Pinched nostrils
   iii. Nasal discharge
   iv. Narrow maxillary arch
   v. Crowded protuding teeth
   vi. Vacant facial expression.
2. *Posterior rhinoscopy examination* – not possible in the young.
3. *Digital palpation* of the nasopharynx is quite painful and better avoided.
4. *X-ray lateral skull* – reveals the adenoids.
5. *Nasal endoscopy* – to visualise the adenoids.
6. *Examination* – under general anaesthesia to visualise or palpate adenoids.

Differential diagnosis:

1. ***Nasal obstruction*** – due to antrochoanal polyp, sinusitis, rhinitis, turbinate hypertrophy etc.
2. ***Dental abnormality*** – giving an appearance of high arched palate and crowded protuding teeth.
3. ***Thornwaldt's cyst*** – which is a remnant of cystic notochord , present as median sac in the midline of the nasopharynx above the upper fibres of superior constrictor muscle, seen in adults .Inflammation of this bursa is called as Thornwaldt's bursitis .

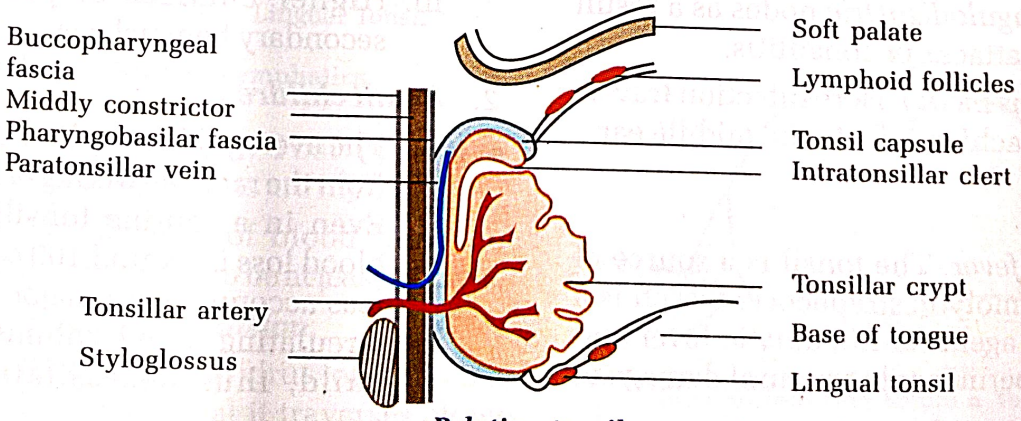
Management:

A) Medical

1. Antibiotics
2. Antihistaminics
3. Local nasal decongestants
4. Improvement of general condition.

B) Surgical

1. Grommet insertion for treatment of deafness
2. Nasal sinus endoscopy for sinusitis
3. Adenoidectomy.



Palatine tonsil

CHAPTER 42
Tonsillectomy and Adenoidectomy

TONSILLECTOMY

Performed along with adenoidectomy around the age group of 4 to 8 years.

Indications:

I. Local

1. *Chronic Tonsillitis.* Patients having 7 attacks of acute tonsillitis in 1 year or 5 attacks for 2 consecutive years or 3 attacks for 3 consecutive years.
2. *Sleep apnoea syndrome.* Enlarged Tonsils and Adenoids causing respiratory obstruction leading to hypoxia, polycythemia and later Cor-Pulmonale.
3. *Chronic enlargement* of the Tonsils causing difficulty in speech, deglutition or respiration.
4. *Tonsillolith.*
5. *Tonsillar cyst.*
6. *Peri-Tonsillar abscess (Quinsy).* After drainage an interval of 6 weeks is required before a tonsillectomy is performed. The chances of recurrent attacks of peritonsillar abscess occurring are high.
7. *Foreign body* like fish bone stuck in the Tonsillar tissue.
8. *Suspected malignancy* of the Tonsil.

II. Focal

1. *Persistent Jugulodigastric* nodes as a result of repeated attacks of Tonsillitis.
2. *Chronic otitis media.* Here infection travels via the eustachian tube to the middle ear.

III. General

1. *Rheumatic fever.* The tonsil is a source of group A hemolytic strepococci which is a recognised agent for Rheumatic fever and can cause permanent structural damage to the heart.
2. *Glomerulonephritis.* Tonsillectomy does not affect established cases but after tonsillectomy the attacks of glomerulonephritis are of lesser frequency and intensity.
3. *Skin conditions* like psoriasis, urticaria and erythema multiforme.
4. *Orthopaedic conditions* like fibrositis and infective arthritis.
5. *Eye conditions* like episcleritis, choroiditis and recurrent conjunctivitis have shown improvement after a Tonsillectomy.
6. *Chronic Bronchitis* and *Asthma* are relieved to a great extent, if they show exacerbation along with an attack of Tonsillitis.
7. *Failure to thrive,* in children.

IV. As an approach for

1. Glossopharyngeal neurectomy done for glossopharyngeal neuralgia.
2. Styloidectomy done for an elongated styloid process causing throat pain and earache.

Contraindictions :

1. ***Acute Tonsillitis***
   - i. Bleeding is more during tonsillectomy.
   - ii. Upper respiratory tract infection spreads via the endotracheal tube leading to lower respiratory tract infection.
   - iii. Higher chances of post-operative secondary hemorrhage.
2. ***Small children***
   - i. The average blood volume is calculated from the ratio of 70 cc/kg of body weight. Even in a routine tonsillectomy the blood loss is around 100 cc. This blood loss accounts for a major reduction of circulating blood volume in a small child, thus necessitating a blood

transfusion. In adults a loss of 100 cc is neglible as compared to the total circulating volume. Therefore children are not taken up for a Tonsillectomy unless they attain a weight of 15kg, when a loss of 100 cc would account for only 10% of the total volume in the child.

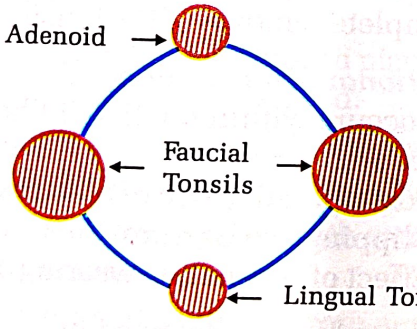
ii. The removal of Tonsils would lead to a compensatory hypertrophy of other lymphoid tissues namely the Lingual Tonsils, which are more difficult to treat.

iii. The function of the tonsils and adenoids is at its peak in a small child (2-3 years) and act as the first line of defence.

iv. All structures are smaller in size, therefore chances of damaging neighbouring tissues is higher.

3. ***Bleeding disorders.*** The tonsil itself being such a vascular tissue, that achieving hemostasis would be impossible.

4. ***Cleft palate*** – Enlarged Tonsils and Adenoids add to the bulk of tissue in the nasopharynx, decreasing the symptoms considerably. Therefore after a Tonsillectomy-adenoidectomy the symptoms of palato-pharyngeal incompetence can worsen.



Waldeyer's Ring of Lymphatics

5. ***Tropical Eosinophilia,*** Post operative pulmonary complications are higher.

6. ***Anemia.*** On account of blood loss, the hemoglobin is required to be at least 11gm%.

7. ***Diabetes and Hypertension.***

8. ***Poliomyelitis.*** The polio virus lodges in the tonsil and after removal it traverses along the exposed glossopharyngeal nerve sheath, thus oral polio leads to a lethal bulbar poliomyelitis.

9. ***Pregnancy*** – If Tonsillectomy has to be done, it can be carried out in the II-Trimester. Menstruation, thought to be associated with a greater bleeding risk from the operative site has been proved to be wrong and is no longer a contraindication to a Tonsillectomy.

Methods of Tonsillectomy

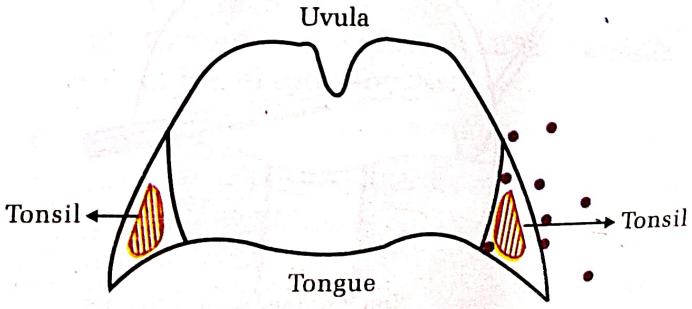
i. Dissection method.
ii. Guillotine.
iii. Cryo Surgery.
iv. Electrocautery.
v. LASER.

Advantages of Dissection Method:

1. Not very time consuming.
2. Can be used for all sizes of tonsils.
3. No damage to neighbouring structures.
4. Complete removal of tonsil ensured.
5. Hemostasis is easy to achieve.

Technique:

1. Atropine is used as a pre-anaesthetic medication, as it
   i. Decreases the apprehension of patients
   ii. Prevents vasovagal attack
   iii. Decreases secretions in the oropharynx
2. Local anaesthesia is preferred. After the throat is sprayed with 4% lignocaine about 5cc of 2% lignocaine with 1 in 2 lac dilution adrenaline is injected into the anterior and posterior pillars.

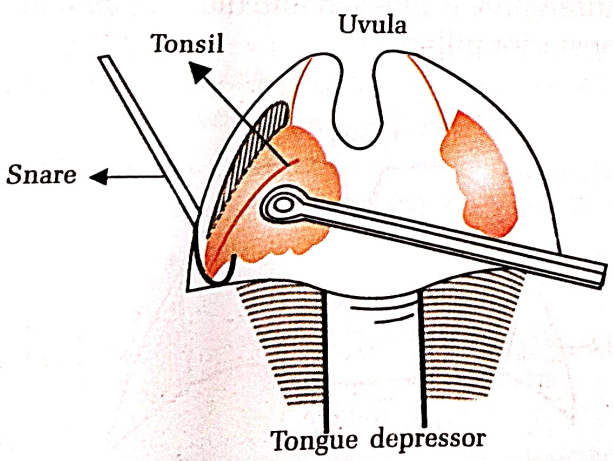


Sites of injection before a Tonsillectomy done under local anaesthesia

The advantages of local anaesthesia are
i. Bleeding is minimal
ii. A good plane of dissection is obtained, therefore no damage to the tonsillar fossa.

In children and unco-operative adults, general anaesthesia is used and throat is packed firmly with a roller gauze

3. The mouth is kept open with either a Doyen's , Boyle-Davis's or Jening's mouth gag with the patient lying supine and the head extended.
4. Incision is made with a tonsillar knife from the base of the anterior pillar to the upper pole of the tonsil.
5. Medial lip of the incision is held with a Tonsil holding or Luc's forceps and lateral lip dissected outwards with a blunt dissector. Gauze strips are introduced into this cleavage to separate the Tonsil from its fossa. This blunt dissection is carried out till the whole Tonsil is separated from the tonsillar fossa excepting the base or pedicle.
6. A snare is fed around the Tonsil and then pedicle is crushed and cut. Now the fossa is packed with a gauze piece to achieve hemostasis.
7. Similar procedure is carried out on the other side.
8. Hemostasis is achieved by taking ties with linen around the pedicle after it has been caught with a tonsillar hemostat, which sloughs off.



Tonsillectomy

Post-Operative

1. Nil by mouth for 4 hrs.
2. Tonsillar position is given, which is
   i. Head low to prevent aspiration of blood as the larynx is now positioned at a higher level than the oral cavity.
   ii. Left lateral position also minimises the chances of aspiration (the right bronchus is a direct continuation of the trachea).
3. Temperature, pulse and respiration are checked every half hourly.
4. Diluted hydrogen peroxide gargles are given after the patient comes fully out of anaesthesia.
5. Diet - avoid solid and hot food.

Complications

Hemorrhage is the only complication of significance and requires management on an emergency basis. It can be divided according to its time of occurrence.

Hemorrhage :

I. ***Primary Hemorrhage***
Occurs on the table itself. The cause of hemorrhage is damage to neighbouring structures like base tongue, uvula, anterior and posterior pillars, or also may be due to incomplete removal of the tonsils.

II. ***Reactionary Hemorrhage***
This occurs within the first 48 hours. The causes are
i. Post-operative rise of blood pressure.
ii. Slipping of a ligature
iii. Effect of adrenaline wearing off

The blood loss is detected by
i. Tachycardia
ii. Hypotension
iii. Rattling noise during breathing
iv. Vomitus of fresh blood.

Post-Tonsillectomy bleeding management :

1. This usually presents in the form of a generalised bleed rather than brisk hemorrhage. The extent of blood loss is

always underestimated as the patient quietly swallows blood in the recovery room (as it is easier to do so, than to spit out the blood).

2. The patient may be hypotensive, hypovolemic and with partial airway obstruction. A stomach full of blood is probable. Preparation includes rehydration with crystalloids through a large bore venflon.
3. Heart rate, blood pressure and bleeding are checked. Two units of blood are kept ready. When the patient is sufficiently hydrated (absence of postural hypotension and tachycardia) then taken to the operation theatre.
4. Any pre-anaesthetic medication or sedating agent is avoided as they cause depression of the respiratory centre.
5. Before inducing the patient, a Ryles tube is passed to empty; the stomach of air and blood.
6. The patient is given head low position and constantly a tonsillar suction is used to clear the oropharynx of blood.
7. Awake endotracheal intubation is performed with pressure on the cricoid ring. This prevents aspiration due to regurgitation of blood from the stomach. (Sellicke's manoeuvre).
8. With the endotracheal tube in place, the cuff is inflated and throat packed.

Bleeding is controlled in the following steps.

i. The clot in the tonsillar fossa is removed.
ii. Pressure application with gauze pieces in the fossa.
iii. Bleeding, if occuring from a particular vessel is caught with a hemostat and ligated.
iv. Agents like silver nitrate or trichloroacetic acid are used in the fossa, in an attempt to stop bleeding.
v. If the above methods to achieve hemostasis fail, suturing of the anterior and posterior pillars is done, keeping gelfoam in the fossa.
vi. As a last resort the external carotid artery is ligated in the neck just after it gives off its first branch, superior thyroid artery
   a) It has to be differentiated from the Internal carotid artery, which has no branches in the neck.
   b) Thrombus distal to the ligation site of the vessel if disloged would enter the superior thyroid artery and not the Internal carotid artery, which would then lead to hemiplegia or blindness.

III. Secondary Hemorrhage

This is hemorrhage occuring after 48 hrs. Generally it occurs around the 4th or 5th Day. The cause of hemorrhage is infection leading to sloughing of tissues. No active surgical management is required and bleeding stops with only

i. Change of Antibiotics
ii. If loss is severe, Blood transfusion
iii. Hydrogen peroxide gargles.

B. Guillotine Method of Tonsillectomy

1. The patient is taken under General Anaesthesia and mouth gag applied.
2. The Guillotine is introduced into the oral cavity pushing the tonsil into its fenestra.
3. On pressing the trigger of the instrument, the blade closes the fenestra cutting off the tonsil.

Disadvantages

i. Incomplete removal of the tonsil
ii. Achieving hemostasis is very difficult.
iii. Damage to neighbouring structures is very high.
iv. Cannot be used for too large, too small or too fibrotic Tonsils.

C. Cryo Surgery

Cryonecrosis of the Tonsils occurs by rapid freezing of its tissues. Liquid Nitrogen is used at – 196° C under a pressure of 22 lbs/sq. inch. The procedure is completed when the Tonsil and adjacent mucosa is covered by frost.

D. Electrocautery

Cauterisation of the Tonsils leads to fibrosis and changes immunity at the cellular level. This prevents repeated attacks of Tonsillitis.

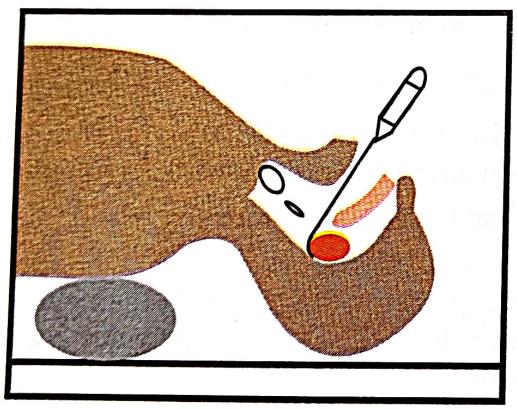
E. LASER

Ablation of Tonsillar tissue by LASER. Used in cases of obstructive sleep apnoea syndrome. Advantages being less bleeding, reduced pain and secondary hemorrhage is rare.

ADENOIDECTOMY

Removal of adenoids is done along with a Tonsillectomy. Chronic infection of the Adenoids would also necessitate its removal. Its signs being

i. Recurrent attacks of Otitis media
ii. Chronic Maxillary Sinusitis
ii. Impaired speech (Nasal)
iv. Mental Lethargy
v. Adenoid facies.



Adenoidectomy

Technique

1. Always performed under general Anaesthesia.
2. Patient lying supine with the head flexed on the neck. This prevents damage to the atlanto-occipital joint, as it abolishes the convexity of upper cervical vertebrae.
3. Finger palpation of adenoids is first done, to :
   i. Confirm the presence and size of adenoid tissue.
   ii. Bring the adenoid tissues in the midline.
   iii. Feel for any abnormal pulsations.
   iv. Ascertain the position of Eustachian tube.
4. A St. Clair Adenoid curette with cage is introduced facing sideways and then turned 90° into the nasopharynx in close contact with the roof and posterior wall of the pharynx. In a single brisk movement the instrument is moved away from the posterior pharyngeal wall to break the adenoid tissues free from the mucosa.
5. If a mass of adenoid tissue is left behind attached to the mucosa, it is grasped with Luc's forceps and cut off with scissors.
6. A nasopharygeal pack of gauze is placed to achieve hemostasis. A thread is always attached to this pack, which prevents the pack from being left inside.

Complications :

1. ***Hemorrhage***
   i. ***Primary***
   Due to damage to the mucosa or incomplete removal of adenoids. The tags of adenoid tissue are removed and postnasal pack is left in place for 10 min.
   ii. ***Reactionary***
   Ice cold packs are placed around the neck for purpose of reflex vasoconstriction. If bleeding persists, a Foley's catheter is introduced via the nose and the bulb is inflated in the nasopharynx for pressure hemostasis. As a last resort, a postnasal pack is inserted with its ends being tied at the columella. This is left in place for 48 hours and the bleeding stops.
   iii. **Secondary**
   This is extremely rare. The patient is given ice packs, sedatives and the antibiotics are changed for control of infection.
2. ***Damage to the eustachian tube orifice*** causing scarring and fibrosis which later

interferes with the tubal function. Later on, this leads to otitis media and conductive hearing loss.

3. ***Subluxation of the Atlanto-occiptal joint.*** Occurs only if the head is placed in a position of extension or if a very forceful action is performed using the currette. The deformity is then treated by traction and an immobilising cervical-collar.

4. ***Hyper-nasality***. Occurs due to removal of a large mass of adenoids causing a sudden increase in the nasopharyngeal space. Also known as Rhinolalia aperta. Enlarged adenoids by virtue of their size facilitate closure of nasopharynx during speech or deglutition. Removal of these adenoids leads to an escape of air during speech, thus imparting a nasal quality to voice. Usually within 6 months there is a compensatory hypertrophy of the palatal muscles, resulting into closure of the nasopharyngeal space during speech or deglutition. Failing this, the treatment constitutes speech therapy exercises which cause the palatal muscles to hypertrophy.

Contraindications

i. Cleft palate
ii. Submucous palate
iii. Bleeding disorders
iv. Anaemia
v. Tropical Eosinophilia

CHAPTER 43

Sleep Apnoea Syndrome

Apnoea – is cessation of airflow at the nostrils and mouth for atleast 10 seconds.

Apnoea may be :

i. *Central* – no airflow as no respiratory effort.

ii. *Obstructive* – no airflow despite respiratory effort

iii. Mixed – combination of central and obstructive apnoea.

Sleep apnoea syndrome is diagnosed when the apnoea index exceeds 5 episodes per hour or 30 episodes over 7 hours. Here we would be dealing with the obstructive form only.

Stridor is noisy high pitched sound resulting from glottic or subglottic obstruction and Stertor is noisy breathing resulting from supraglottic obstruction.

Pathophysiology

There is a partial obstruction to the airway at the supraglottic, pharyngeal or nasal level. Prolonged obstruction to airway results in hypoxia leading to polycythemia and later Cor Pulmonale.

Etiology

1. **Age** – highest incidence around 50 years.
2. **Sex** – common in males
3. **Alcohol** – excessive consumption and snoring are frequently associated.
4. **Obesity** – responsible for obstruction to airway results in hypoxia leading to polycythemia and later Cor Pulmonale.
5. **Drugs** – sedatives, antihistaminics, tranquillizers etc.

Clinical features

1. Loud snoring
2. Abnormal movements during sleep
3. Personality changes
4. Difficulty in concentration
5. Excessive daytime sleeping
6. Morning headaches.
7. Nocturnal enuresis.

Investigation

1. Fibreoptic endoscopy – of nose, pharynx and larynx., to find the level of obstruction.
2. X-ray skull, paranasal sinuses, neck and chest.
3. CT Scan to detect soft tissue or bony obstructions.
4. Pulmonary function tests would reveal hypoxia.
5. Rhinomanometry measures the intra nasal pressures.
6. Sleep latency time.
7. Nocturnal polysomnography consists of a battery of tests conducted during sleep to detect apnoea .

Differential diagnosis

1. Narcolepsy
2. Chronic obstructive lung disease
3. Congestive cardiac failure
4. Hypoglycemia
5. Severe anaemia
6. Hypothyroidism

Predisposing factors

1. Large tonsils (Faucial and lingual)
2. Adenoids
3. Septal deformities
4. Allergy
5. Nasal polyps
6. Tumours.

Management:

I. Medical

1. Weight reduction
2. Alcohol consumption stopped

3. Avoiding drugs precipitating sleep apnoea
4. CPAP-Continuous Positive Airway Pressure (Low-flow oxygen therapy)
5. Prosthesis to hold tongue forward.

II. Surgical

1. Nasal surgery – turbinate reduction, septoplasty, polypectomy etc.
2. Tonsil – adenoid removal
3. Uvulopalatopharyngoplasty done by laser .
4. Mandibular advancement
5. Tracheostomy in extreme cases.

CPAP Machine

CHAPTER 44
Pharyngitis

Causes:

I. Acute
- i. Non-specific acute pharyngitis
- ii. Diphtheria
- iii. Thrush
- iv. Herpes
- v. Infectious mononucleosis
- vi. Actinomycosis
- vii. Agranulocytosis

II. Chronic
- i. Non-specific chronic pharyngitis
- ii. Tuberculosis
- iii. Leprosy
- iv. Scleroma
- v. Syphilis
- vi. Sarcoidosis
- vii. Keratosis pharyngis

NON-SPECIFIC ACUTE PHARYNGITIS

A common condition, that frequently may be associated with acute tonsillitis.

Etiology
1. Associated with common cold
2. Irritation from fumes
3. Severe digestive disturbances.
4. Organisms – streptococci, pneumococci, staphylococci etc.

Pathology
There is congestion of mucous membrane of the posterior pharyngeal wall and the lymphoid nodules stand out prominently, enlarged and darker (red) than the surrounding.

Stages
1. Acute simple pharyngitis
2. Infective pharyngitis
3. Membranous pharyngitis

Symptoms
1. Sore throat-raw sensation
2. Pain on swallowing, which is less when swallowing food than saliva.
3. Neck swelling
4. Severe dysphagia
5. Toxaemia.

Signs
1. Congestion of posterior pharyngeal wall
2. Enlargement of lymphoid nodules
3. Neck nodes are enlarged and tender.

Differential diagnosis
The other causes of acute pharyngitis.

Management
1. Rest
2. Analgesics and antibiotics
3. Plenty of oral fluids
4. Warm saline gargles.

NON-SPECIFIC CHRONIC PHARYNGITIS

It is a common condition with a wide range of etiological agents.

Etiology
1. Allergic rhinitis
2. Chronic sinusitis
3. Following tonsillectomy
4. Polluted and dusty environment
5. Chronic bronchitis and bronchiectasis
6. Atrophic rhinitis
7. Tropical eosinophilia
8. Alcohol and smoking habits
9. Paan and tobacco chewing
10. Dental sepsis.

Types
1. Catarrhal pharyngitis
2. Hypertrophic granular pharyngitis
3. Atrophic pharyngitis

Symptoms

1. Throat irritation
2. Cough
3. Blood stained sputum
4. Vocal fatigue.

Signs

1. Congestion of posterior pharyngeal wall.
2. Dilated blood vessels over the pharyngeal mucosa.
3. Active mucus discharge.
4. Follicles or cysts may be seen.

Investigations

1. Hemogram – anemia
2. X-ray paranasal sinuses – sinusitis
3. X-ray chest – tuberculosis or bronchiectasis
4. Throat swab – smear, culture and antibiotic sensitivity (SCABS).

Treatment

1. General condition is improved.
2. Causes if detected,are treated.
3. Gargles may be soothing.
4. Steam inhalation.
5. Silver nitrate cautery of the granules or follicles on the posterior pharyngeal wall.
6. Electrocautery in resistant cases.
7. LASER.

CHAPTER 45

Deep Neck Infections

QUINSY (PERITONSILLAR ABSCESS)

Quinsy occurs as a complication of acute tonsillitis, when infection passes through the tonsillar capsule and lies within the superior constrictior muscle where it produces cellulitis and then an abscess.

Etiology

1. **Age** – occurs in adults.
2. **Sex** – seen more often in males.
3. **Causative organism** – as in acute tonsillitis.
4. **Foreign body** – embedded into the tonsillar tissue may lead to quinsy.
5. **Tonsillar tag** – may also lead to quinsy.

Pathology

Following an attack of acute tonsillitis the opening of the intratonsillar cleft (crypta magna) becomes closed due to fibrosis. Later, with a fresh attack of acute tonsillitis infection spreads into the tonsillar tissue to break its capsule and later cause an abscess.

Symptoms

1. Severe throat pain on one side which aggravates on swallowing.
2. Inability to open the mouth following spasm of pterygoid muscles.
3. Fever, upto 104 F.
4. Salivation resulting from throat irritation.
5. Foeter due to secondary infections.
6. Earache is unilateral and reffered..
7. Muffled and thick speech (Hot potato speech)

Signs

1. Trismus depends upon the severity of infection.
2. Congested anterior pillar
3. Tonsil is pushed medially and downwards by the abscess.
4. Uvula is edematous and pushed towards the normal(opposite) tonsil.
5. Foul odour
6. Marked cervical lymphadenitis.
7. Torticollis due to reflex spasm of neck muscles.

Differential diagnosis of unilateral tonsillar enlargement

1. Quinsy
2. Tonsillolith
3. Malignancy of the Tonsil
4. Tonsillar cyst.

Complications

1. Parapharyngeal abscess
2. Jugular vein thrombosis
3. Septicemia
4. Laryngeal edema.
5. Lung abscess or pneumonitis due to aspiration of pus.
6. Haemorrhage from carotid artery or jugular vein.

Management:

A) Conservative

i. Systemic antibiotics in high doses.
ii. Analgesics and anti-inflammatory drugs.
iii. Bed rest.
iv. Plenty of oral fluids.

B) Surgical:

Drainage is to be performed once the abscess has formed.

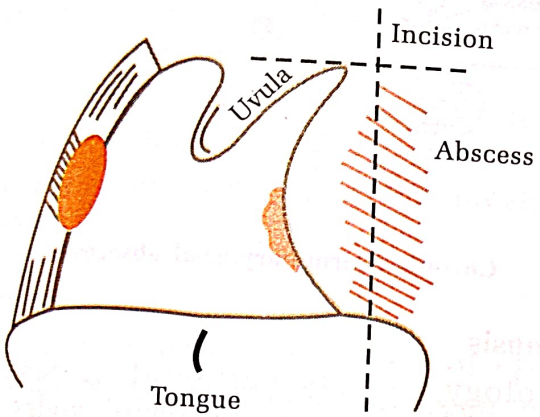
i. Local anaesthesia is preferred as there is a possibility of rupture of the abscess during intubation for general anaesthesia.
ii. Incision is made by a quinsy knife at the point of maximum bulge or at the

intersection of a line from the base of uvula and one passing along the anterior pillar.

iii. Following rupture of the abscess, gargles with hydrogen peroxide is given.

iv. Tonsillectomy is done after a period of 4-6 weeks to prevent recurrent attacks of quinsy.

v. Quinsy – Tonsillectomy is to be avoided as :
   a) Rupture of the abscess and aspiration during intubation could occur.
   b) Bleeding is excessive due to infection.
   c) Chances of thrombo-embolism are high.



Site for drainage of (L) Quinsy

PARAPHARYNGEAL ABSCESS

It is a suppurative infection of the parapharyngeal space seen more frequently in adolesents and adults.

Etiology

1) **Dental** – lower wisdom tooth
2) **Tonsil** – quinsy
3) **Ear** – Bezold's abscess
4) **Trauma** – neck injuries
5) **Infections** – from parotid, retropharyngeal or submaxillary spaces.

Clinical features

1. Throat pain is very severe.
2. Trismus due to spasm of pterygoid muscles.
3. Pyrexia is high grade.
4. Neck swelling following the abscess and lymph node involvement.
5. Pharyngeal wall and tonsil are pushed medially.

Complications

1. Laryngeal edema
2. Thrombophlebitis of internal jugular vein.
3. Septicemia
4. Mediastinitis following spread along the carotid sheath.
5. Spontaneous rupture of carotid artery.

Differential diagnosis

1. Quinsy
2. Retropharyngeal abscess
3. Tumours of pharynx
4. Vascular aneurysms.

Management

1. Systemic antibiotics in high doses
2. Analgesics and anti-inflammatory agents
3. Incision and drainage done, when the abscess is pointing. This can be done intraorally or by an external incision.
4. Tracheostomy may be required.

ACUTE RETROPHARNYGEAL ABSCESS

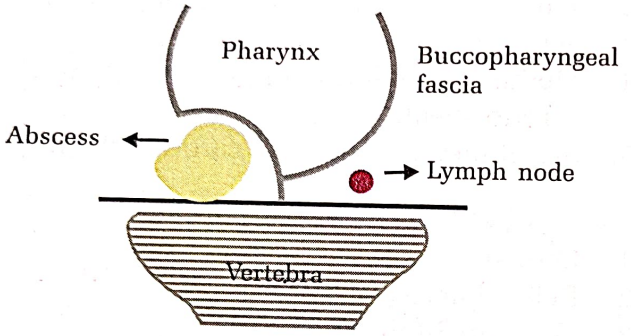
It is suppuration of the retropharyngeal lymph nodes in the space of Gilette.

Etiology

1. **Age** – occurs in children, mostly under one year of age.
2. **Sex** – boys are more often affected.
3. **Source** – oropharyngeal or nasopharyngeal infection. Usually tonsillar or dental infection is present.
4. **Trauma** – following rupture of the pharyngeal wall by a sharp foreign body.
5. **Mastoid abscess** – very rarely tracks down.
6. **Causative organisms** – streptococci and staphylococci.

Clinical features

1. Difficulty in breathing and sucking
2. Croupy cough
3. Nasal obstruction
4. Neck stiffness or torticollis
5. Neck swelling due to lymphadenitis
6. Pyrexia
7. Posterior pharyngeal bulge on oral cavity examination.
8. Toxaemia.



Acute Retropharyngeal abscess

Radiology

Lateral neck view shows soft tissue swelling in the retrophayngeal space. A fluid level may also be seen.

Complications

1. Laryngeal edema
2. Spontaneous rupture of the abscess and aspiration
3. Dehydration due to decreased intake.

Management

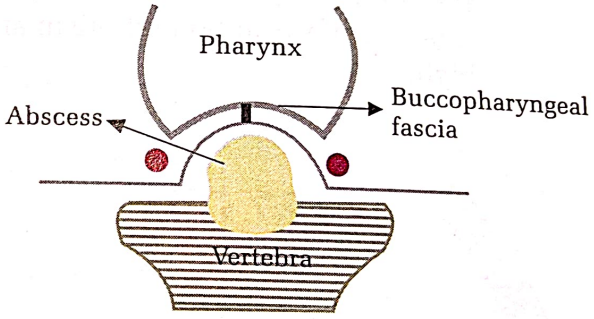
1. Antibiotics and anti-inflammatory drugs.
2. Fluid intake is maintained.
3. Incision and drainage is done via transoral route,. No anaesthesia in used. Infants are wrapped in a towel and given head low position before the drainage. Availability of suction is a must for fear of aspiration of the pus. General anaesthesia is avoided, as fear of rupturing the abscess during intubation is present.
4. Tracheostomy may be required.

CHRONIC RETROPHARYNGEAL ABSCESS

It occurs following tuberculous caries of the cervical vertebrae spreading behind the prevertebral fascia or infection of retropharyngeal lymph nodes.

Clinical features

1. Slight dysphagia
2. Sore throat and cough
3. Enlarged painless lymph nodes
4. Cold abscess may be present on the posterior pharyngeal wall
5. Restricted neck movements.



Chronic Retropharyngeal absecess

Diagnosis

Radiology reveals vertebral disease or calcification of tuberculous lymph nodes. An infected branchial cyst is to be excluded.

Management

1. Incision of abscess is made in front of the sternomastoid muscles externally and intraoral incision is contraindicated.
2. Spinal caries have to be treated.
3. Anti-tuberculous treatment is essential.

CHAPTER 46

Tumours of the Pharynx

NASOPHARYNGEAL ANGIOFIBROMA

It is a benign vascular tumour, occuring in the nasopharynx in adolescent males.

Theories of formation

1. From periosteum of base of the skull under the influence of endocrine hormones.
2. Remnants of craniopharyngeal duct.
3. From the fascia basalis.
4. Due to deficiency of androgens or overactivity of estrogens
5. Ectopic vascular tissue, growing due to pitutary gland disturbance.
6. Tumour cells arising from the maxillary artery.

Pathology:

Gross sites of attachment are roof of the nasopharynx, medial pterygoid plate, posterior border of hard palate and posterior part of vomer.

Microscopically consists of blood vessels and fibrous tissue.

Clinical features

1. Nasal block.
2. Epistaxis is profuse.
3. Underdeveloped secondary sexual characters.
4. Rhinolalia clausa.
5. Anterior and posterior rhinoscopy reveal the tumour.
6. Eustachian tube block leading to deafness.
7. Tumour spread would cause proptosis, cheek swelling or broadening of the nose. (Frog - face deformity).

Investigations

Biopsy is never taken as it may cause massive bleeding

1. X-ray of the nasopharynx shows bowing of the posterior wall of antrum (Angiofibroma filling the pterygopalatine fossa) referred to as 'Antral sign'. The x-ray of fronto-occipital view shows area of bony destruction with invasion of sphenoid sinus.
2. Angiography is performed to determine the extent and vascularity of the tumour, along with the size and location of its feeding vessels.
3. CT Scan.

Differential diagnosis

1. Antrochoanal polyp
2. Hypertrophied adenoids
3. Chordoma
4. Post nasal tumours

Treatment

1. Surgical removal of tumour is required. The approaches available are transpalatal, lateral rhinotomy or transmaxillary.
2. Cryosurgery.
3. Radiotherapy.
4. Hormone therapy.

Prognosis

A residual tumour is seen more often than its recurrence after surgical removal. Regression of the tumour occurs with age.

CARCINOMA OF THE NASOPHARYNX

Squamous cell carcinoma is the commonest of the malignant nasopharyngeal tumours.

Etiology

1. **Age** – occurs after 40 years.
2. **Sex** – commoner in males.
3. **Diet** – dry salted fish containing carcinogenic nitrosamine.
4. **Chronic sinusitis and nasopharyngitis.**
5. **Epstein** – Barr virus.
6. **Endocrine** – males having high estrogen levels.
7. **Environmental** – exposure to smoke from incense or wood.

Pathology

Gross – exophytic, ulcerative or infiltrative growth. Fossa of Rosenmuller is a common site of initiation of tumour.

Spread

1. Direct
2. Lymphatic
3. Blood borne.

Clinical features

1. Metastasis of neck nodes
2. Local invasion (Trotter's triad)
   i. Conductive deafness
   ii. Immobility of homolateral soft palate
   iii. Pain on the side of the head.
3. Nasal obstruction
4. Epistaxis
5. Symptoms of Invasion
   i. Strabismus due to VI nerve palsy.
   ii. Enopthalmus from orbital invasion.
   iii. Jugular foramen syndrome causing paresis of IX, X and XI cranial nerves.
6. Pain and headache signifies tumour erosion of the base skull and its surrounding structures.

Diagnosis

1. Anterior rhinoscopy-unilateral nasal mass.
2. Posterior rhinoscopy-growth seen occupying fossa of Rosenmuller.
3. Nasopharyngoscopy done under general anaesthesia.
4. Radiology includes X-ray skull and CT Scan to see for extent of the tumour.
5. Biopsy is a must for confirmation of diagnosis.
6. Elevated antibody titres to the Epstein-Barr virus

Management:

Radiotherapy – is the treatment of choice as these tumours are radiosensitive . Surgical removal of the growth is not possible due to its involvement with the base skull. Radiotherapy is given with a dose of 6000 rads over a period of six weeks.

Chemotherapy – Used in advanced nodal disease (>6cm), where radiotherapy is ineffective. Adjuvant chemotherapy is effective in reducing local and regional relapses of the tumour.

Surgery – Limited to radical neck dissection and control of post–radiation cervical metastasis.

CHAPTER 47
Miscellaneous Diseases of the Pharynx

HYPOPHARYNGEAL POUCH (DIVERTICULUM)

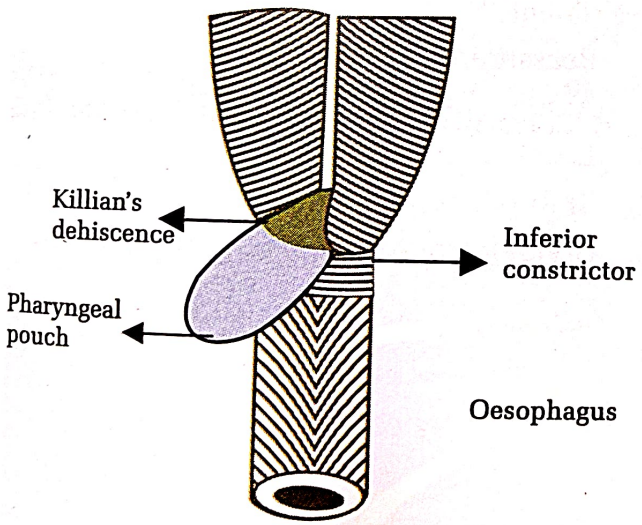
It is a herniation of the pharyngeal mucosa through Killian's dehiscence.

Etiology

1. Age – seen in elderly.
2. Sex – common in males.
3. Familial – high incidence in certain families.

The various factors responsible are :

1. Tonic spasm of the cricopharyngeus muscle prevents the downward passage of bolus and the weak area (Killian's dehiscence) gives way.
2. Neuromuscular inco-ordination leading to a delay in the relaxation of cricopharyngeal sphincter at the end of second stage of swallowing.



Killian's dehiscence

3. Some patients take large mouthfull of food which remains in the pharynx even after the sphincter has closed.
4. Spasm of the cricopharyngeus may be caused by reflux oesophagitis.

Pathology

The pouch is composed of mucosa and fibrous tissue only. Gradually the opening of the pouch becomes a more direct continuation of the pharynx and oesophagus.

Clinical features

1. Dysphagia which is of long standing.
2. Regurgation of undigested food.
3. Neck swelling.
4. Cough.
5. Emaciation.

Diagnosis

Radiography with barium swallow shows a retort shaped swelling, which may extend into the mediastinum.

Treatment

1. Very small pouches require no treatment.
2. Small pouches are treated by dividing circular fibres of the cricopharyngeal sphincter by an external route.
3. Endoscopic division of the wall between the lumen of the pharynx and that of oesophagus may be performed by a diathermy.
4. Large pouches are excised by an external route.
5. Oesophago – diverticular anastomosis is done for very large pouches present in the superior mediastinum.

GLOBUS HYSTERICUS

It is a sensation of lump in the throat.

Etiology

1. Age – occurs in adults.
2. Sex – females are commonly affected.
3. Psychological – factors, usually fear of cancer is the underlying cause.

Clinical features

Feeling of 'lump in the throat' is present. There is no true dysphagia for either solids or liquids.

Differential diagnosis

1. Cricopharyngeal spasm
2. Hypopharyngeal pouch
3. Cervical osteophytes
4. Post-cricoid malignancy
5. Hiatus hernia
6. Thyroid adenoma.

Investigations

Barium swallow and oesophagoscopy is normal.

Treatment

1. Exclusion of an organic disease is essential.
2. Tranquillizers.
3. Reassurance.
4. Psychiatric help.

KERATOSIS PHARYNGIS

Clinical features

White or yellow projection appears on the surface of tonsils, pharyngeal wall and base of tongue. It is seen in young adults. Although symptomless, the appearance alarms the patient.

Pathology

They are composed of horny desquamated epithelium and these projections cannot be wiped off. There is no inflammation and the cause is unknown.

Treatment

1. Improvement of oral hygiene and general health.
2. The condition usually regresses spontaneously and so best left alone. Tonsillectomy is not justified.

STYLOHYOID SYNDROME (Eagle's syndrome or Styalgia)

Clinical features

Sharp pain is felt in the involved fossa which is lateralised to one side at hyoid or suprahyoid level and pain is referred to the affected side of ear. The symptoms persist for years and are aggravated by coughing, sneezing, swallowing or straining. Tenderness is present above the attachment of the stylohyoid ligament. It can be palpated in the tonsillar fossa which can be diagnosed by X-ray of styloid process and confirmed by the relief of pain after local anaesthetic injection in the fossa .

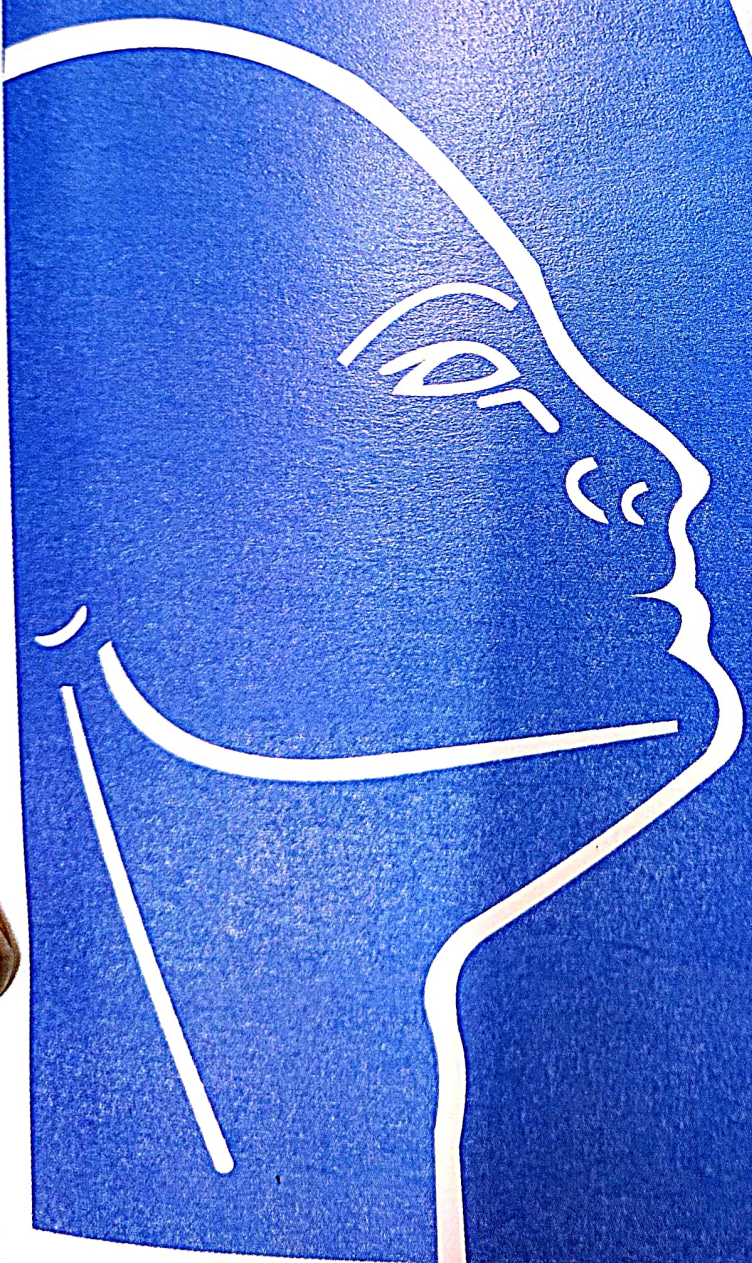
The exact etiology and pathology are not known. It is due to calcified or elongated styloid process.

Treatment

1. Reassurance.
2. Analgesics and anti-inflammatory drugs.
3. Local steroid injections.
4. Tranquillizers.
5. Styloidectomy following tonsillectomy.

SECTION - V

OESOPHAGUS



CHAPTER 48

Anatomy of the Oesophagus

The Oesophagus is a muscular tube approximately 25 cm. long in an adult.

Wall

1. Muscular layer
2. Submucosal layer
3. Mucosal layer

Blood supply

i. Cervical part – inferior thyroid artery
ii. Thoracic part – descending thoracic aorta
iv. Abdominal part – left gastric artery.

Venous drainage

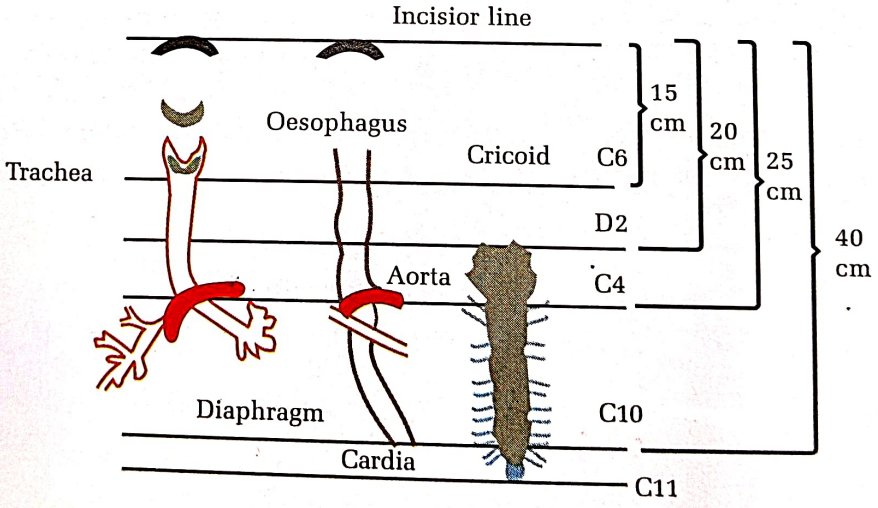
i. Cervical part – inferior thyroid vein.
ii. Thoracic part – azygous vein
iii. Abdominal part – left gastric vein.

Nerve supply

1. Vagus nerve (parasympathetic)
2. Cervical sympathetic.

Lymphatic drainage

1. Cervical part – lower cervical nodes.
2. Thoracic part – posterior mediastinal nodes.
3. Abdominal part – left gastric nodes.



Relations of Oesophagus

Examination

1. Barium swallow
2. Oesophagoscopy.

CHAPTER 49

Oesophagoscopy

It is endoscopic examination of the Oesophagus.

Indications:

I. ***Diagnostic:***
1. Investigating a case of dysphagia
2. Suspected foreign body
3. An unknown primary for a neck swelling
4. Vocal cord palsy
5. Biopsy to rule out malignancy, as in Achalasia Cardia.

II. ***Therapeutic:***
1. Foreign body removal
2. Dilatation of strictures and webs
3. Removal of benign tumours eg. polyps
4. Tracheo-oesophageal fistula repair
5. Introduction of feeding tube in Carcinoma
6. Oesophageal intubation
7. Injection of sclerosing agents in oesophageal varices.

Contraindications

1. Generalised Medical disorders
2. Trismus
3. Cervical spine disorders
4. Aortic aneurysm
5. History of Oesophageal perforation.

Technique

1. Done under General Anaesthesia.
2. Patient lying supine in Boyce's position.
3. The scope is passed from the right angle of the mouth along the posterior pharyngeal wall behind the endotracheal tube.
4. A horizontal slit like structure is seen at about 15 cm from the incisors, which is the cricopharynx.
5. When the cricophyarynx relaxes on its own, the scope is passed into the Oesophagus where transmitted aortic pulsations are seen and then later a constriction at the site of the left bronchus.
6. At about 40 cms from the incisors the Cardia is reached which is confirmed by:
   i. Sudden change of the mucosal pattern
   ii. Regurgitation of gastric juice
   iii. Entry into a large cavity
   iv. Cessation of respiratory movements
7. Even on removal of the oesophagoscope, the mucosa is watched closely. This is to detect a hidden lesion seen due to the unfolding of oesophageal mucosa seen on removal of the scope.

Complications

1. Anaesthesia.
2. Damage to lip, teeth, arytenoids and vocal cords.
3. Oesophageal perforation.

   The management being
   i. Nil by mouth
   ii. Ryles tube passed on table
   iii. Intravenous fluids
   iv. Antibiotics and Analgesics
   iv. Monitoring of vital parameters.

CHAPTER 50
Dysphagia

Dysphagia is difficulty in swallowing while painful swallowing is termed as odynophagia.

Causes:

I. Mouth

1. Stomatitis
2. Cleft palate
3. Palatal palsy
4. Oronasal fistula
5. Xerostomia following radiotherapy
6. Mikulicz disease.

II. Oral Cavity

1. Trismus
2. Fracture of mandible
3. Temporomandibular joint disorders
4. Carcinoma of jaw
5. Lingual abscess
6. Ludwig's Angina
7. Paralysis of tongue
8. Tumours of tongue
9. Total glossectomy

III. Pharynx

1. Acute Tonsillitis
2. Quinsy
3. Malignancy
4. Retropharyngeal abscess
5. Foreign body impaction
6. Laryngeal oedema
7. Spasmodic conditions like tetanus, rabies etc.
8. Paralysis because of bulbar palsy, diphtheria, cerebrovascular accidents etc.

IV. Oesophagus

A) In the lumen:

1. Foreign bodies like coins, dentures etc.
2. Benign or malignant tumours
3. Strictures.

B) In the wall:

1. Benign strictures
   a) Reflux oesophagitis
   b) Corrosive injury
   c) Traumatic injuries
2. Malignant strictures
3. Plummer – Vinson syndrome
4. Myasthenia gravis
5. Globus hystericus
6. Tetanus
7. Bulbar palsy
8. Benign tumours
9. Achalasia cardia
10. Diverticulum
11. Hiatus- hernia
12. Tracheo-oesophageal fistula

C) Outside the wall:

a) Cervical

1. Pharyngeal pouch
2. Thyroid lesions e.g. enlargement, Hashimoto's thyroiditis,malignancy.
3. Cervical osteophytes
4. Lymph node enlargement

b) Thoracic

1. Retrosternal goitre
2. Cardiomegaly
3. Pericardial effusion
4. Mediastinal tumours
5. Bronchogenic carcinoma
6. Dysphagia lusoria. (vascular rings).
7. Aortic aneurysm

Clinical features

1. Age :

a) *Plummer – Vinson Syndrome* occurs around 40 years of age
b) *Malignancy of oesophagus* occurs after 40 years of age.
c) *Achalasia cardia* is found in the age group of 20-40 years.

2. **Sex:**
   Plummer –Vinson syndrome occurs in females only.

3. **Past History:**
   a) *Poliomyelitis or diphtheria* – oesophageal paralysis.
   b) *Corrosive swallowing and instrumentation* – benign stricture.
   c) *Anemia, glossitis and stomatitis* – Plummer Vinson syndrome.
   d) *Psychiatric disorders* – Globus hystericus.

4. **Symptoms:**
   a) *Sudden onset* – foreign body or acute oesophagitis
   b) *Slow onset* – cardiospasm, malignancy or stricture formation.
   c) *Dysphagia for solids* – malignancy or stricture of the oesophagus
   d) *Dysphagia for liquids* – cardiospasm or paralytic lesions.
   e) *Retrosternal pain* – reflex oesophagitis
   f) *Regurgitation* – cardiospasm.
   g) *Progressive* – malignancy

5. **Signs:**
   Carcinoma of the oesophagus may be suspected with neck swelling, weight loss, hepatomegaly or ascites.

Investigations

1. *Haemogram*- Plummer Vinson syndrome.
2. *X-ray chest* - mediastinal mass, cardiovacular or pulmonary diseases.
3. *X-ray neck* – post cricoid tumour, retropharyngeal lesion or osteophytes.
4. *Barium swallow* – malignancy, strictures, diverticulae, achalasia cardia or hiatus hernia.
5. *Oesophagoscopy* – direct visualisation and biopsy.
6. *Manometric and pH studies* – acid reflux or motility disorders.
7. *Bronchoscopy* – bronchogenic carcinoma.
8. *Thyroid scan* – malignancy.
9. *Cardiac catheterisation* – vascular anomalies.

CHAPTER 51
Diseases of the Oesophagus

CARCINOMA OF THE OESOPHAGUS

Management of Oesophageal malignancies becomes difficult because of its anatomical location and delayed manifestation of its symptoms.

Identification of the specimen of Oesophagus:

i. Hollow tubular organ
ii. Absence of serosa
iii. Longitudinal folds
iv. Presence of adjacent structures eg. stomach, aorta, lungs etc.

Etiology

i. Age – Seen above the age of 50 years
ii. Sex – Common in males (M:F = 4:1)
iii. Predisposing conditions
- Achalasia Cardia
- Barrett's oesophagus
- Corrosive strictures
- Diverticulosis
- Plummer Vinson syndrome
- Scleroderma

Gross Pathology

Sites:

i. Upper third - 20%
ii. Middle third - 50%
iii. Lower third - 30%

Types

i. Ulcerative
ii. Cauliflower
ii. Annular

Microscopic Pathology

i. Squamous Cell Carcinoma (upper or middle third of oesophagus).
ii. Adenocarcinoma (usually lower third, as arising from the gastric mucosa).
iii. Anaplastic carcinoma.

Symptoms

1. Dysphagia – initially for solids and later for both solids and liquids.
2. Aspiration
3. Cough with expectoration
4. Hematemesis
5. Cachexia
6. Pain
7. Left supracalvicular swelling (Virchow's node)

Spread

1. Local – due to absence of serosa, the spread occurs very fast.
2. Lymphatics – Submucosal lymphatics lead to vertical spread of tumour giving rise to satellite nodules. Resection of the tumour is complete only if 10 cm of the specimen is removed from either side of the malignant growth (to distinguish whether an ante mortem specimen).
3. Blood - Liver
Lung
Bone

Management:

1. *Upper third of oesophagus – Radiotherapy* As important structures are present in the mediastinum, which cannot be resected.
2. *Lower third of oesophagus – Surgery.* Adenocarcinoma is radio-resistant and replacement of excised oesophagus is done easily by stomach.
3. *Middle third oesophagus* – Choice of surgeon or patient and also depending upon the facilities available.

Cause of death:

1. Cachexia
2. Hematemesis
3. Metastasis
4. Mediastinitis
5. Aspiration.

PLUMMER-VINSON'S (PATERSON- KELLY) SYNDROME

A syndrome of gradually increasing dysphagia to solids, hypochromic microcytic anaemia with certain changes in the epithelium.

Etiology

1. Age – occurs around the age of 40 years
2. Sex – exclusively in females
3. Iron deficiency anemia

Pathology

Characterised by thining of the mucosa of the upper alimentary tract with disappearance of rete pegs and a reduction of glycogen content in the cells. The mucosa later on undergoes fibrosis and there is web formation.

Clinical features

1. Increasing dysphagia to solids
2. Glossitis (atrophy of the papillae)
3. Angular stomatitis
4. Koilonychia (spoon shaped) or brittle finger nails
5. Pruritis
6. Splenomegaly.

Investigations

1. Barium swallow may show a filling defect at the upper end of the oesophagus with dilated hypopharynx.
2. Oesophagoscopy may reveal a web or a stricture.
3. Hemoglobin is low, demonstrating hypochromic microcytic anemia.
4. Gastric analysis for achlorhydria.

Treatment

1. Iron and vitamin supplements are given in high doses.
2. Oesophageal strictures are dealt by dilatation.
3. Patients are kept under observation to check for early malignant changes.

ACHALASIA CARDIA (CARDIOSPASM)

It is a failure of the cardia to relax for the passage of food. There is degeneration of the ganglion cells in Auerbach's plexus resulting into failure of relaxation of the lower oesophageal sphincter in response to a swallow.

Etiology

1. Due to degeneration in the neurones of both vagal trunks and in the cells of their central nuclei.
2. Degeneration first occurs in utero and is due to temporary ischemia of this area during rotation of the gut.
3. Frequently associated with Chaga's disease, which is an infection with Trypanosoma cruzi.

Clinical features

1. Seen between 30 to 60 years of age.
2. Commonly occurs in males.
3. Epigastric discomfort.
4. Dysphagia with temporary remissions.
5. Regurgitation of frothy mucopus,mixed with swallowed food and fluids.
6. Later, Pulmonary complications due to aspiration are common.

Diagnosis

1. Radiology – findings are typical. (refer page no. 242)
2. Oesophagoscopy, done not only to confirm the diagnosis but also to exclude the possibility of a co-existing carcinoma.

Treatment

1. Drugs – Octyl nitrate, probanthine etc.
2. Dilatation of oesophagus.
3. Surgical intervention, by incising the muscle (not through the mucous membrane) at the cardio – oesophageal juntion.

HIATUS HERNIA

There are two types of herniation of the stomach into the thorax.

Types:

1. **Para-oesophageal hernia**

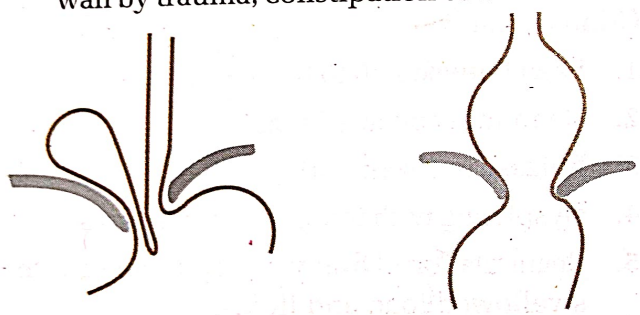
   Here there is herniation of a part of the stomach and peritoneum through the hiatus of the diaphragm alongside the oesophagus. The cardia of the stomach remains in its normal position.

   Cause – Congenital.

2. **Sliding hernia**

   Here the stomach slides upwards through the diaphragm and part of it remains within the thorax, with the cardia being within the thorax.

   Cause – obesity, compression of abdominal wall by trauma, constipation etc.



Para oesophageal type **Sliding type**

Clinical features

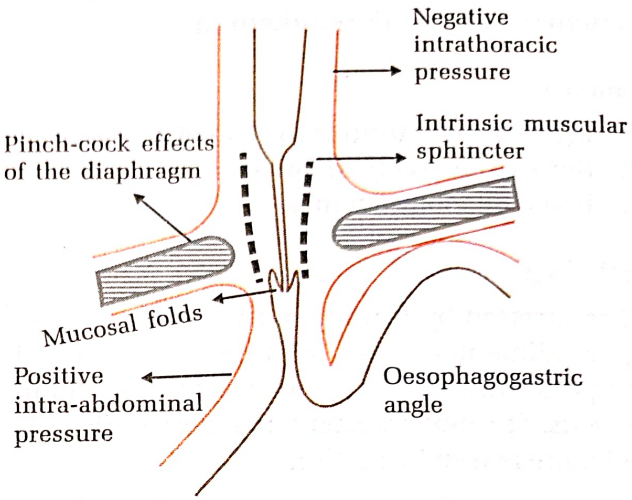
1. Reflux oesophagitis causing heartburn.
2. Bleeding (hematemesis or malena).
3. Breathlessness.
4. Mass in the chest.

Diagnosis

1. Barium swallow
2. Oesophagoscopy. Malignancy must be excluded.

Treatment

1. Antacids
2. Reduction of weight.
3. Frequent small meals.
4. Propped up position with pillows during sleep.
5. Surgical repair of the diaphragmatic opening and replacement of the viscus.
   Done only after failure of medical treatment.



Factors involved in the prevention of oesophagogastric reflux

Dysphagia lusoria

An aberrant right subclavian artery compressing the oesophagus resulting into dysphagia .

The vessel arises from the descending aortic arch and passes obliquely upward towards the right arm, passing behind the esophagus instead of ventral to the trachea.

In 15 % of patients it passes between the oesophagus and trachea. It represents an abnormality of the 4th branchial arch.

The incidence is 1 % of population and is highest in congenital heart disease patients. The compressive symptoms will appear in 10 % of patients, not found until adulthood.

The symptoms are more likely to occur when the left and right carotid arteries originate either from a common trunk or very close together, so that trachea and oesophagus are prevented from deviating forward between the carotid arteries to escape the posterior compression.

Clinical features

Symptoms

In infants - Dysphagia (Commonly symptoms appear once diet begins to include solid food)

Regurgitation and aspiration

Stridor (Tracheal compression)

Cyanosis (Improves on hyper-extension of neck)

Adults – Mild dysphagia with sticking sensation at the level of xiphoid process. They present in later life, primarily as the atheroscleorosis develops along with progressive oesophageal dysmotility.

Diagnosis

1) ***Radiography*** – Barium study - Will show an indentation of the posterior esophageal wall near the level of the aortic arch at the 3rd and 4th thoracic vertebrae.
2) ***Endoscopy*** – Sometimes reveal pulsatile compression, mainly helpful in excluding other causes. The pressure with the tip of the endoscope may obliterate the right radial pulse.
3) ***Angiography*** –Useful only when surgery is indicated.

Treatment – Usually supportive, particularly in adults.

Surgical correction is necessary only when the problem interferes with nutrition and is causing significant aspiration.

Surgical ligation of aberrant subclavian artery is done, provided that collateral circulation is adequate. Otherwise a reanastomosis to the ascending aorta is necessary.

SECTION - VI

LARYNX

CHAPTER 52

Anatomy of the Larynx

The larynx is situated in the midline of the neck at the meeting of the digestive and respiratory tracts, from the level of the third to the sixth cervical vertebrae. It consists of a framework of cartilages, connected by ligaments and membranes, lined by mucous membrane and moved by muscles.

Laryngeal Cartilages – form the framework of the larynx.

a) **Unpaired cartilages**
1. Thyroid
2. Cricoid
3. Epiglottis

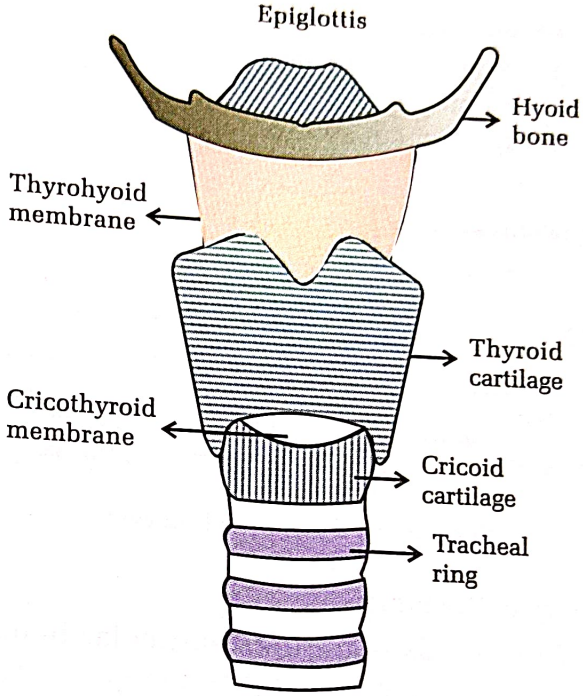
b) **Paired cartilages**
1. Arytenoid
2. Corniculate
3. Cuneiform

Laryngeal joints – are present on either sides of the larynx.
1. Cricothyroid.
2. Cricoarytenoid.

Laryngeal ligaments and membranes:

A) **Intrinsic** – uniting the cartilages of the larynx to one another.
1. Elastic membrane of larynx
2. Conus Elasticus
3. Thyro – epiglottic ligament.

B) **Extrinsic** – uniting cartilages of the larynx to skeletal structures outside the larynx.
1. Thyrohyoid membrane
2. Median thyroid ligament
3. Lateral thyroid ligament
4. Cricotracheal membrane
5. Hyo-epiglottic ligament.



Laryngeal cartilages and Hyoid bone

Laryngeal muscles

A) ***Intrinsic*** - between one laryngeal cartilage and another.

I. **Abductor of the vocal cord**
Posterior crico-arytenoid

II. ***Adductors of the vocal cord***
1. Lateral crico-arytenoid
2. Transverse portion of interarytenoid
3. External portion of thyro-arytenoid

III. **Tensors of the vocal cord**
1. Cricothyroid
2. Internal portion of thyro-arytenoid (vocalis).

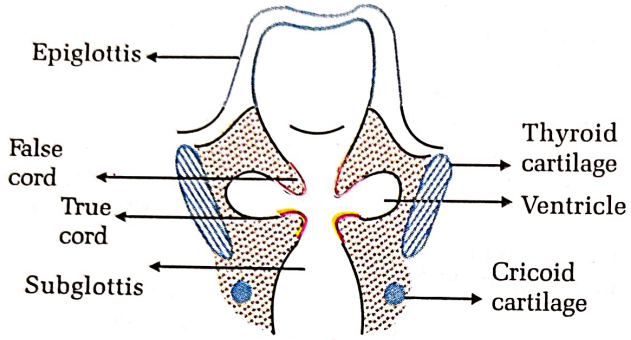
IV. **Opening the laryngeal inlet**
Thyro-epiglottic

V. **Closing the laryngeal inlet**
1. Oblique portion of interarytenoid
2. Ary-epiglottic

C. **Extrinsic** - between the larynx and neighbouring structures.

I. **Strap muscles**
1. Sternothyroid
2. Thyrohyoid

II. **Pharyngeal muscles**
1. Stylopharyngeus
2. Palatopharyngeus
3. Inferior constrictor



Coronal section through Larynx

Cavity of the larynx:

1. **False cords** – are the ventricular brands formed by the mucous membrane covering the ventricular ligaments.
2. **True cords** – project further into the cavity and lie at a lower level than the false cords. Anteriorly it is attached in the midline to the posterior surface of the thyroid cartilage and posteriorly to the arytenoid cartilages.
3. **Ventricle of the larynx** – is a recess between the false and true vocal cords.

Mucous membrane of the larynx – lines the whole cavity. It is continuous above with that of the mouth and laryngopharynx, and below with that of the trachea.

Blood supply

1. Superior thyroid artery
2. Inferior thyroid artery
3. Cricothyroid artery

Nerve Supply

1. ***Superior laryngeal nerve.***
   a) Internal branch supplies larynx above the level of the true vocal cords (sensory).
   b) External branch supplies the cricothyroid muscle (motor).
2. ***Recurrent laryngeal nerve.***
   It runs a longer course on the left side as it turns around the arch of aorta, while on the right, it turns around the subclavian artery.
   a) Motor branch supplies all intrinsic muscles of the larynx except cricothyroid muscle.
   b) Sensory branch supplies the larynx below the level of true vocal cords.

Lymphatic drainage:

1. **Supraglottic** – above the true vocal cords, drain into
   i. Pre-epiglottic nodes
   ii. Upper deep cervical nodes.
2. **Glottic** – the true vocal cords have practically no lymphatic vessels, hence malignant tumours limited to them do not spread easily.
3. **Subglottic** – below the true vocal cords, drain into
   i. Prelaryngeal and pretracheal nodes
   ii. Lower deep cervical nodes.

	Infant Larynx	Adult Larynx
1. Position	Higher up	4th to 6th cervical vertebra
2. Sex	No difference	Male larynx is bigger
3. Epiglottis	Folded and narrow	Leaf like
4. Submucosa	Abundant	Less
5. Cartilage	Soft	Rigid
6. Narrowest area	Subglottis	Glottis
7. Adam's apple	Not felt	Prominent
8. Nerve supply	Extensive	Less

CHAPTER 53

Functions of the Larynx

I. Protection of the lower air passages

This is the most important function and the earliest one to develop. Mechanisms involved here are

1. Closure of the laryngeal inlet.
2. Closure of the glottis.
3. Cessation of respiration.
4. Cough reflex.

II. Phonation

Voice is produced by the vibration of the vocal cords. The sound produced, is imparted quality by the resonating chambers of the mouth, pharynx, nose and paranasal sinuses.

III. Respiration

The reflex adjustments of the glottic aperture contribute to the regulation of acid-base balance in the body by influencing the carbon-dioxide tension of blood.

IV. Fixation of the chest

Before performing strenous work, the glottis is closed and this fixes the chest. This helps during climbing, defaecation, childbirth etc.

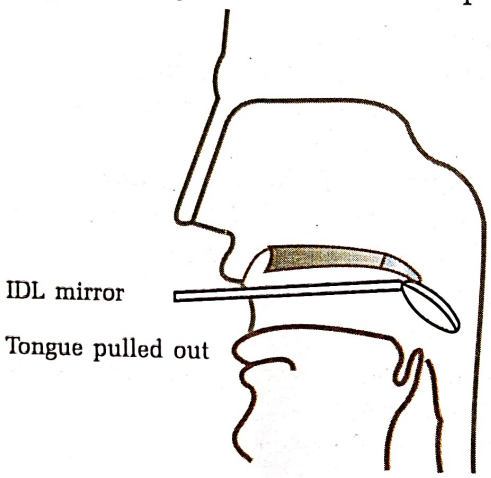
CHAPTER 54
Examination of the Larynx

EXTERNAL EXAMINATION

i. ***Inspection*** – of larynx to note its position, deviation from the midline and its movements during deglutition.

ii. ***Palpation*** – is done to detect tenderness (perichondritis) and broadening of the larynx (malignancy).

Thyrocervical crackle is present on moving the larynx against the vertebral column. This grating sound (sensation) is absent in post-cricoid carcinoma, retro-pharyngeal abscess and foreign bodies at the cricopharynx.



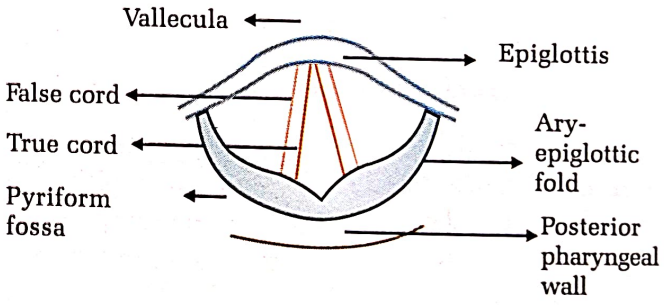
Indirect Laryngoscopy

INDIRECT LARYNGOSCOPY

It is a simple OPD procedure which can yield plenty of information. Good illumination, gentleness of the examiner and relaxation on the part of the patient are essentials for success.

Procedure:

1. The indirect laryngoscopy mirror is warmed on a spirit lamp or dipped in hot water to prevent fogging of the mirror by condensation of vapour from the patients breath (not for sterilisation).
2. The patient sits upright with the head bent slightly forwards. If the patient has a very active gag reflex, use of an anaesthetic spray (4% lignocaine) is helpful.
3. The patient protudes his tongue which is held by a gauze piece between the thumb and middle finger of the left hand of the examiner. The index finger is used to raise the upper lip.
4. The patient is asked to breathe easily and steadily from the mouth.
5. The indirect laryngoscopy mirror is held like a pen in the right hand and placed in the oral cavity, resting on the soft palate without touching the posterior pharyngeal wall.
6. If all structures are not seen, the same routine is to be followed each time.



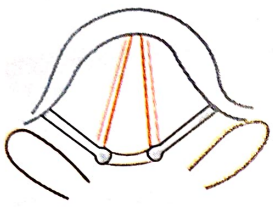
Indirect Laryngoscopy

Structures seen from above downwards

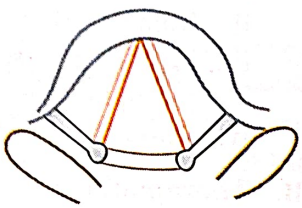
i Base of the tongue
ii. Vallecula on either side of glosso-epiglottic fold
iii. Epiglottis
iv. Ary-epiglottic folds
v. Arytenoid cartilages
vi. False cords
vii. True cords
viii. Upper rings of trachea may be seen
ix. Pyriform fossa

Movements of the Vocal cords

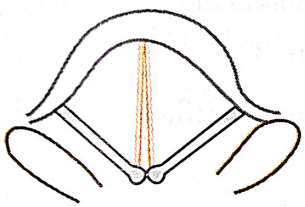
1. *Quiet respiration* – the cords lie moderately separated and equidistant from the midline. On inspiration they move outwards and on expiration they move in again, but do not reach the midline.
2. *Forced inspiration* – the cords move out till they are close to the sides of the larynx.
3. *Phonation* – the cords come together in the midline.



Moderate abduction in quiet respiration



Full abduction in deep breathing



Adduction on phonation

Vocal cord movement

DIRECT LARYNGOSCOPY

A direct view of the larynx is necessary due to the following drawbacks of indirect laryngoscopic examination.

i. Fore-shortening of the vertical axis to one-third of its distance.
ii. Mirror image shows an anterior-posterior reversal of the structures.
iii. False sensation of depth of the larynx.
iv. Feeling that vocal cord is flat. This is felt due to the shadow of the overhanging ventricular band.
v. Due to angulation of the mirror, the size of lesions at the anterior commisure appear smaller in size.
vi. Overhang of the epiglottis, hides many a lesions.
vii. The anterior commissure, ventricle, subglottis cannot be seen.

Indications:

I. Diagnostic

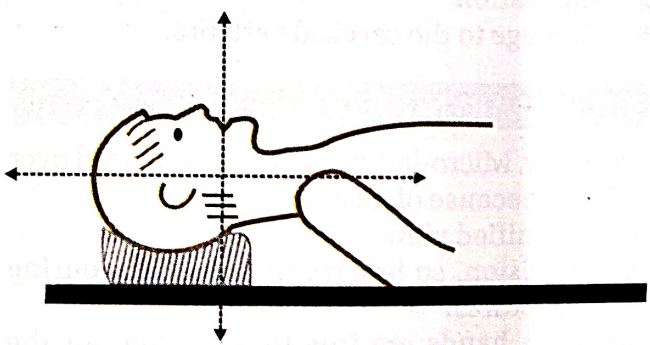
i. Investigations for a case having laryngeal symptoms.
ii. In upper Aerodigestive tract malignancies, to determine the extent and for biopsy of lesions.
iii Vocal cord paralysis.
iv. For assessing laryngeal trauma.
v. Unknown primary site for neck swellings, as a part of panendoscopy.
vi. Suspected Foreign body.

II. Therapeutic

i. Foreign body removal.
ii. Excising benign tumours eg. papilloma.
iii. Treatment of Premalignant conditions eg. leucopakia.
iv. Insertion of Laryngeal stent.
v. Teflon paste-injection in cases of adductor palsy.
vi. Dilatation for Subglottic stenosis.
vii. Where Ultrasound, LASER or Cryo is to be used on the larynx.

Contraindications

1. Medical disorders where General Anaesthesia cannot be given.
2. Cervical spine disorders.
3. Trismus.
4. Stridor.



Normally the oral and the laryngeal/pharyngeal axes lie perpendicular to each other

On lying supine the Oral cavity axis and the Pharyngeal - Laryngeal axis lie perpendicular to each other. The patient is given **Boyce's position** : flexion of the cervical spine and extension at the atlanto-occipital joint. Now the vertical axis becomes more horizontal and the horizontal (Pharyngeal/Laryngeal) axis more vertical. Only after achieving this position, the axes lie in one plane and then can a rigid instrument be passed.

Technique

1. Preferrably done under General Anaesthesia.
2. Patient lying supine, Boyce's position is given.
3. A Direct Laryngoscope is introduced from the right angle of the mouth, taking care not to damage the upper incisors and the lip.
4. The structures seen are the Right Tonsil, Base of Tongue, Vallecula between the Glossoepiglottic folds, Epiglottis, the anaesthetic endotracheal tube is lifted up to see the Arytenoids, Pyriform fossa, Lateral Pharyngeal wall, False cords, Ventricle of the Larynx and the True cords.

Complications

1. Of Anaesthesia
2. Damage to lip, teeth, tongue, arytenoids or the vocal cords.
3. Vasovagal shock, due to excessive manipulation at the laryngeal inlet.
4. Laryngeal edema
5. Aspiration
6. Damage to the cervical vertebra.

MICRO-LARYNGOSCOPY

Presently, Micro-laryngoscopy is preferred over a direct because of its advantages of

i. Magnified view
ii. Precision, so less trauma to neighbouring structures.
iii. Both hands are free for carrying out the procedure as the scope is fixed to a chest piece.
iv. Binocular vision, therefore exact depth of the lesion is obtained.
v. The surgeon is away from the toxic anaesthesia gases.
vi. Side tube or Video attachment allows simultaneous teaching for students, as well as documentation is possible.

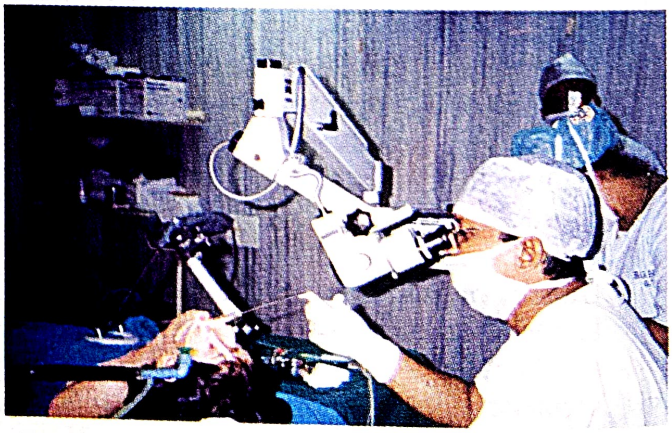
Indications:

A. **Diagnostic**
   i. Investigation of Laryngeal symptoms
   ii. Suspected Foreign body.
   iii. Biopsy in suspected cases of Laryngeal malignancies.
   iv. Assesment of Laryngeal trauma.

B. **Therapeutic**
   i. Removal of benign lesions eg. polyp or vocal nodule.
   ii. Excision of premalignant lesions eg. leucoplakia or carcinoma in situ.
   iii. Teflon paste injection for adductor palsy.
   iv. Arytenoid cartilage removal in abductor palsy.
   v. For the use of Ultrasound, LASER or Cryo.

Technique

1. The microlaryngoscope is introduced under General Anaesthesia (A small endotracheal tube is used or the tube would itself conceal the view of the larynx).
2. The larynx is visualised after the scope is fixed to a chest piece and a lens of 400 mm length is attached to the microscope.



Micro-laryngoscopy using a chest piece

Complications

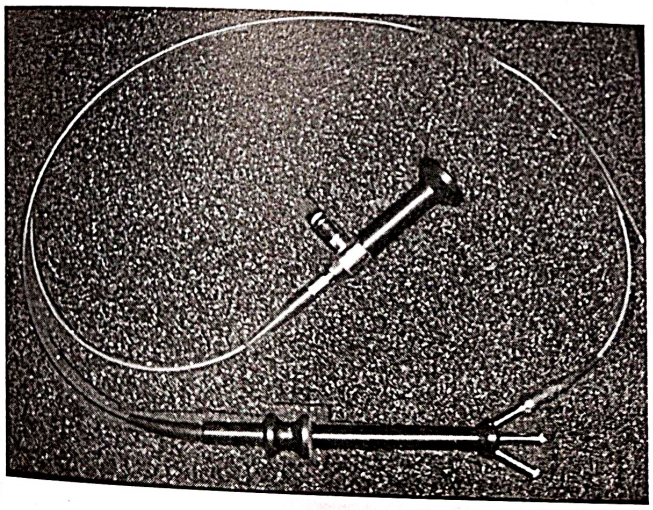
1. Damage to structures, may occur similar as with a Direct Laryngoscope.
2. Cardiac problems occuring during the procedure are higher, due to
   i. Hypoxia caused by a small endotracheal tube.
   ii. Chest piece preventing complete expansion of the lungs.
   iii. Excessive manipulation of the Larynx leads to vagal stimulation.

FIBRE-OPTIC FLEXIBLE LARYNGOSCOPY

The flexible fibre-optic laryngoscope is passed through the nose and nasopharynx to reach the laryngeal inlet.

Indications

1. Difficult indirect laryngoscopic examination.
2. Used in children.
3. Trismus, (where other forms of laryngeal examination are not possible)
4. Unconscious patient.



Paediatric Flexible Fibre-Optic Scope

Advantages

1. It is an OPD procedure
2. Movements of the vocal cords are visualised.
3. Photography is possible.
4. Magnified view of the larynx obtained.

RADIOGRAPHY

1. Soft tissue radiograph.
2. Contrast laryngogram
3. Tomogram
4. CT Scan.

STROBOSCOPY

The rapid vibration of the vocal cords during phonation cannot be noted during an indirect laryngoscopic examination. Here, an interrupted light source is used during the indirect laryngoscopic examination.

By adjusting the speed of the flash of light, the movements of the vocal cords can be slowed or even 'frozen'. Valuable information can thus be obtained in a patient who has an obviously abnormal voice but whose larynx appears normal on an indirect laryngoscopy examination.

CHAPTER 55
Laryngitis

Types:

I. Acute
1. Laryngitis
2. Laryngotracheobronchitis
3. Epiglottitis
4. Diphtheric laryngitis.

II. Chronic
1. Non-specific laryngitis
2. Hyperkeratosis
3. Scleroma
4. Leprosy
5. Syphilis
6. Tuberculosis.

ACUTE LARYNGITIS

It is an acute superficial inflammation of the laryngeal mucous membrane.

Etiology

1. ***Infection*** – as part of a generalised upper respiratory infection or as localised laryngitis.
2. ***Trauma*** – due to vocal abuse or any endoscopic manipulation.
3. ***Irritation*** – from inhaled fumes or gases including smoking.

Pathology

1. Congestion of the mucosa
2. Edema of the mucosal surface
3. Mucopurulent exudate
4. Perichondritis.

Clinical features

1. Hoarseness of voice
2. Discomfort in the throat
3. Dyspnoea due to the mucosal edema
4. Painful swallowing
5. Dry irritative cough
6. Malaise and fever
7. Vocal cords appear congested with sticky mucus.

Complications

1. Spread to tracheobronchial tree
2. Children are prone to dyspnoea and stridor
3. Perichondritis, may develop in severe cases

Treatment

1. Local
- i. Voice rest
- ii. Steam inhalations
- iii. Lozenges
- iv. Fomentation of the neck
- v. Linctus for irritating cough.

General

1. Antibiotics are helpful in the early cases
2. Avoidance of allergens
3. Anti-inflammatory drugs to reduce congestion and edema
4. Steroids to reduce the laryngeal edema
5. Oxygen in cases of respiratory distress.

ACUTE EPIGLOTITIS

A special form of acute laryngitis where the inflammatory changes affect mainly the loosely attached mucosa of the epiglottis.

Clinical features

1. Dyspnoea, may be severe in children
2. Pain on swallowing is a feature in adults
3. Stridor in children - Inspiratory
4. Sorethroat.

Signs – Fever,
Cervical lymphadenopathy.

On any condition one should not examine the pharynx, as it may cause Asphyxia .

Investigations – Complete blood count - (Leucocytosis).

X-ray Neck - *"Thumb sign"* - Edematous epiglottis appears enlarged.

Treatment

1. Constant supervision till dyspnoea and stridor subsides
2. Antibiotics
3. Systemic steroids
4. Tracheostomy may be required.
5. Oxygen
6. Plenty of fluids
7. Endotracheal intubation or tracheostomy
8. Suctioning to remove secretions via the tracheostomy opening or by bronchoscopy.

ACUTE LARYNGOTRACHEOBRONCHITIS

A condition affecting infants and young children when their resistance is low and it is characterised by generalised edema of the tracheobronchial tree.

Etiology

Causative organism is usually hemolytic streptococcus but it may be caused by a virus.

Clinical features

1. Croupy cough
2. Hoarseness of voice
3. Exudation
4. Laryngeal edema
5. Atelectasis.

Differential diagnosis

1. Acute laryngitis
2. Foreign body bronchus
3. Bronchopneumonia
4. Diphtheria.

Treatment

1. Rest
2. Systemic antibiotics
3. Systemic steroids
4. Humidification of inspired air

DIPHTHERIC LARYNGITIS

It is usually an extension of faucial diphtheria but is difficult to diagnose when primarily laryngeal.

Etiology

1. Age – Occurs in children below 10 years of age.
2. Organism – Corynebacterium diphtheria.

Clinical features

1. Cough – is hoarse and croupy in nature
2. Stridor – is inspiratory
3. Weak and rapid pulse
4. Fever
5. Greyish-white membrane, appears on the affected parts and on separation causes bleeding.

Differential diagnosis

1. Acute laryngotracheobronchitis
2. Inhaled foreign body
3. Acute laryngitis
4. Allergic laryngitis
5. Laryngismus stridulus
6. Laryngeal papillomas.

Investigation

Microscopic examination of a laryngeal swab would confirm cornyebacterium diphtheriae.

	Diphtheric laryngitis	Acute laryngotracheobronchitis
i. History	Exposure to diphtheria	Upper respiratory infection
ii. Fever	Low	High
iii. Toxemia	Less	More
iv. Stridor	Inspiratory	Expiratory
v. Throat	Diphtheric patch may be seen	Acute tonsillitis
vi. Chest	Clear	Rales and rhonchi
vii. Laryngoscopy	Diphtheric membrane	Marked congestion and edema of larynx

Treatment

1. Antitoxin injection is given ranging from 20,000 to 1,00,000 units according to age.
2. Antibiotics – penicillin.
3. Steroids to reduce the edema.
4. Oxygen.
5. Intubation or tracheostomy to overcome laryngeal obstruction.

CHRONIC NON-SPECIFIC LARYNGITIS

Etiology:

1. Faulty use of voice – vocal abuse or overuse.
2. Infection – in the teeth, tonsils or sinuses.
3. Dust or fumes inhalation.
4. Alcohol or tobacco excesses.
5. Generalised disorders – gout, rheumatic arthritis etc.

Symptoms

1. Hoarseness, more in the morning.
2. Dry irritating cough
3. Raw sensation in the throat
4. Hawking, a frequent desire to clear the throat.

Signs

1. Vocal cord congestion
2. Hyperplasia of the cords
3. Edema
4. Sticky mucus around the cords
5. The larynx is always affected bilaterally and symmetrically.

Treatment

1. Voice rest
2. Steam inhalations
3. Smoking and alcohol to be avoided
4. Linctus codeine for cough
5. Environmental change may help
6. Stripping of the vocal cords is performed in resistance cases.

DIFFERENTIAL DIAGNOSIS – Of laryngeal lesions.

Laryngeal Lesions	Tuberculosis	Syphilis	Malignancy	Non specific Chronic laryngitis
1. Pain	Severe	Absent	May be present	Insignificant
2. Swallowing	Painful	Normal	Dysphagia later	Normal
3. Emaciation	Present	Absent	Present	Absent
4. Voice	Weak aphonic	Strong hoarse	Hoarse	Hoarse
5. Lesions	Post 1/3 affected pale or mouse nibbled appearance	Ant 1/3 involved - Deep ulcers	Everted edges	Congestion
6. Radiograph of chest	Pulmonary tuberculosis	Normal	Secondaries in the late stages	Normal
7. Sputum	A.F.B. Present	Normal	Negative	Negative
8. VDRL	Negative	Positive	Negative	Negative

Biopsy gives the diagnosis.

CHAPTER 56

Paralysis of the larynx

Laryngeal paralysis

I. **Organic:**

A) **Motor** – Unilateral or bilateral

1. Incomplete - only abductors are paralysed
2. Complete - both abductors and adductors are paralysed.

B) **Sensory** – unilateral or bilateral

II. **Functional.**

MOTOR PARALYSIS

Etiology:

1. **Lesions affecting the Vagus (X) nerve**

A) **Supranuclear** – rare, as larynx has bilateral representation in the motor area of cortex.

B) **Nuclear** –

i. Hemorrhage or thrombosis of posterior inferior cerebellar artery.
ii. Disseminated sclerosis
iii. Tumours
iv. Diphtheria
v. Bulbar poliomyelitis.

C) **Infranuclear** – intracranial

i. Meningitis
ii. Posterior fossa tumours
iii. Base skull fractures.

2. **Lesions affecting recurrent laryngeal nerve**

A) Left recurrent laryngeal nerve:

i. Aneurysm of aorta
ii. Oesophageal malignancy
iii. Bronchogenic carcinoma
iv. Apical tuberculosis
v. Mediastinal tumours
vi. Thoracic surgery
vi. Thyroid malignancy
vii. Metastatic neck nodes.

B) Right recurrent laryngeal nerve:

i. Thyroid surgery
ii. Aneurysm of right subclavian artery
iii. Apical tuberculosis
iv. Apical lung carcinoma
v. Thyroid malignancy

3. **Generalised disorders:**

Peripheral neuritis from

i. Diabetes
ii. Lead poisoning
iii. Syphilis
iv. Diphtheria
v. Viral infections.

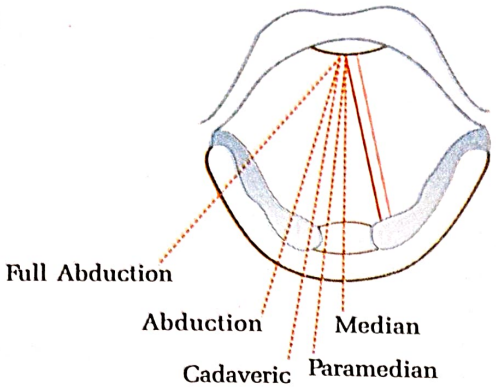
4. **Idiopathic.**

Clinical features

1. Recurrent laryngeal nerve lesion.
   a) Unilateral – voice and respiration unaffected.
   b) Bilateral – the voice is good but slight exertion causes dyspnoea as cords are in the paramedian position.
2. Superior laryngeal nerve lesion.
   The voice is rough and tires easily but respiration is unaffected.

Investigations

1. ESR – tuberculosis
2. VDRL – syphilis
3. Blood sugar – diabetes
4. Cardiovascular examination – mitral stenosis or aortic aneurysm
5. Neurological examination – intracranial lesions
6. X-ray chest – tuberculosis, cardiomegaly etc.
7. Barium swallow – oesophageal malignancy
8. Bronchoscopy and oesophagoscopy to detect malignancy



Positions of the vocal cord

9. Fine needle aspiration cytology – of neck nodes or thyroid to diagnose malignancy.
10. CT Scan – intracranial and mediastinal lesions.

Management:

I. Recurrent laryngeal nerve paralysis

A) **Unilateral:**

1. Asymptomatic patient needs no specific treatment.
2. Cause, if found is treated.
3. Speech therapy to improve voice.
4. Teflon paste injection done to push the paralysed cord towards the midline.

B) **Bilateral:**

1. Asymptomatic patient needs no specific treatment.
2. Cause, if detected is treated.
3. Endotracheal intubation is done, if dyspnoea is present.
4. Tracheostomy may be required.
5. Arytenoid cartiage excision is done to create an opening for respiration.
6. The vocal cord can be sutured laterally to give the patient an adequate airway. (However, these operations result in loss of speech as midline approximation of the vocal cords is not possible).

II. Superior laryngeal nerve palsy

1. The cause, if detected is treated.
2. Ryles tube feeding to avoid aspiration.
3. Tracheostomy, may be required.

FUNCTIONAL PARALYSIS (Hysterical-Aphonia)

Functional paralysis of the adductors occurs mostly in emotionally unstable individuals particularly young women.

Clinical features

1. Aphonia
2. Laryngeal paraesthesia
3. Cords fail to meet on attempting phonation
4. Cough is normal.

Treatment

1. Persuasion
2. Psychotherapy
3. Speech therapy

SEMON'S LAW

It states that in a course of gradually advancing organic lesions of recurrent laryngeal nerve, the abductors are affected before the adductor muscles and in case of recovery the adductor muscles recover before the abductors; and paralysis of purely adductor type is functional in origin.

Stages:

1. Initially abductors are affected and vocal cord moves to the midline.
2. Additionally spasm of the adductors and therefore the cord is immobilised in the median position.
3. Adductors get affected and then vocal cord lies in the cadaveric position.
4. Next is compensation by the other (normal) vocal cord.

Postulates :

1. Due to their late development with evolution, the abductors are more vulnerable to disease.
2. Mass of adductors is much more than the muscle mass of abductors.
3. The nerve fibres of the abductors are situated more externally to the adductors, so more prone to external pressure.
4. The adductors being protective in function, are the last to get affected and the first to recover.

CHAPTER 57

Hoarseness of the Voice

A hoarse voice is one which is harsh, low pitched and rough as compared to the normal voice.

Etiology – Causes:

1. **Congenital**
   - i. Laryngeal web
   - ii. Laryngomalacia
   - iii. Dermoid
   - iv. Cystic hygroma
   - v. Hemangioma
2. **Inflammatory**
   - i. Acute laryngitis
   - ii. Diphtheria
   - iii. Viral
   - iv. Singer's nodule
   - v. Vocal polyp
   - vi. Scleroma
   - vii. Leprosy
   - viii. Chronic laryngitis
   - ix. Tuberculosis.
   - x. Atrophic laryngitis.
3. **Traumatic**
   - i. Inhaled foreign body
   - ii. Intubation injury
   - iii. Post coughing, shouting etc.
   - v. Strangulation
   - vi. Submucosal haemorrhage
4. **Neoplastic**
   - i. Papilloma
   - ii. Squamous cell carcinoma
   - iii. Fibroma
   - vii. Chondroma
   - viii. Lipoma
   - ix. Leiomyoma
   - x. Haemangioma
   - xi. Carcinoma
   - xii. Angiofibroma
   - xiii. Amyloid tumour
   - xiv. Laryngocele
5. **Paralytic**
   - i. Organic paralysis of the vocal cord
   - ii. Functional aphonia
6. **Miscellaneous**
   - i. Cyst
   - ii. Myxoedema
   - iii. Crico-arytenoid joint fixation
   - iv. Gout
   - v. Dysphonia plica ventricularis

Investigation:

1. *Detailed history* –Vocal overuse or abuse is to be ruled out. Hoarseness persisting for more than 3 weeks requires to be examined in detail.
2. *Complete ENT examination* – includes an indirect laryngoscopy to rule out local causes.
3. *X-ray neck and chest*-tumours and cardiovascular or respiratory conditions.
4. Direct laryngoscopy and Micro-laryngoscopy- to take biopsy.
5. *CT Scan*- for tumours.
6. *Bronchoscopy*- brochogenic carcinoma.
7. *Oesophagoscopy*- malignancy.

Miscellaneous conditions :

Adductor spasmodic dysphonia
(Spastic dysphonia / Mogiphonia / Phonic Spasms / Laryngeal Stuttering / Laryngeal cramp)

It is a disorder of phonation characterized by a strained, creaking and choking vocal attack with a tense and squeezed vocal sound, accompanied by tension in accessory phonatory musculature.

This unpredictable, intermittent over-adduction of vocal cords is seen around 40 years of life.

Patients prone for stuttering

- Parkinsonism
- Torticollis
- Hemifacial spasm
- Blepharospasm.
- Tremor syndrome.

Clinical features

Symptoms - Patient tries to speak but cannot speak completely. The voice is described as tight, effortful, low pitched, harsh, squeezed or struggle strained.

To overcome this patient will try throat clearing, falsetto voice usage, facial grimacing and does some forms of facial expressions.

The symptoms are aggravated by emotion, fatigue and anxiety states. Temporary improvement seen in

- Heavy sedation
- High alcohol intake
- Post anaesthesia

Signs –IDL shows spasms of vocal cords (both true and false cords), seen mainly at the end of forced expiration or during phonation. In severe cases, one can see tight adduction of the false vocal folds during phonation.

Management

As such speech therapy does not relieve the condition, but helps the patient to understand the condition better.

Psychotherapy advocated.

Surgery – Section of Recurrent laryngeal nerve, done to reduce the force of glottic closure (the strained strangled voice will be replaced by slight breathing voice, due to give way of paralysed fold on attempted phonation), followed by speech therapy to improve voice quality (like tone clarity, pitch control and vocal loudness).

1) Secondary endoscopic procedure to debulk through a cordotomy incision the paraglottic musculature which lateralises paralysed vocal cord further.
2) Injection of botulinum toxin (paralyse the thyroarytenoid muscle temporarily –6 weeks to 9 months).

ABDUCTOR SPASMODIC DYSPHONIA
(Breathy spasmodic dysphonia)

It is characterised by sudden episodes of breathiness or unvoiced segments and is precipitated by unvoiced/voiceless consonants. (such as s, h, f, t, k)

IDL reveals - intermittent abduction of vocal cords on phonation.

Managed by Speech therapy.

LARYNGISMUS STRIDULUS

It is the episodic spasms of the larynx seen in 2 to 3 years old child.

This occurs secondary to tetany which develop due to insufficient vitamin D or calcium intake or excessive loss of calcium into the urine.

Clinical features - Indrawing of chest , crowing inspiratory stridor (due to episodes of glottic spasm), temporary apnoea, cyanosis associated with convulsions, twitching and carpopedal spasms.

The symptoms appear due to increased irritability of the nervous system.

Chvostek's and Trousseau's signs are positive and serum calcium levels are very low.

Management

1) Dietary supplementation of Calcium and Vitamin D.
2) Injection of Calcium gluconate given intravenously slowly.
3) Self limiting disease. The disease regresses once parathyroid functions becomes normal.

LARYNGEAL SPASM IN ADULTS

The spasm of organic etiology seen in adults, is divided into two types.

1) Tonic Spasms
2) Clonic spasms (Pharyngo-laryngeal nystagmus)

Tonic Spasms

It present as severe spasm of larynx with pain.

Causes

1) Tabes dorsalis
2) Syringomyelia
3) Multiple sclerosis
4) Progressive bulbar palsy
5) Thoracic malignancies. (Due to irritation of recurrent laryngeal nerve)
6) Adhesions of Pleura
7) Aortic aneurysm
8) Tetanus
9) Rabies

Diagnosis – Usually the larynx is normal but one should suspect it on the basis of underlying disease.

IDL sometimes shows inappropriate adduction of the vocal folds / cords during attempted breathing.

Management – Treat the underlying condition.

Clonic spasms. (Pharyngo-laryngeal nystagmus) In this there will be involuntary, rhythmic, jerking movements seen in the laryngo-pharynx, tongue, palate and facial muscles. The respiratory stridor is always due to central nervous origin.

Causes

1) Multiple sclerosis.
2) Encephalitis.
3) Meningitis.
4) Tumours of cerebellar and frontal lobe.
5) Cerebrovascular insufficiency or hemorrhage.

Treatment – Treat the underlying condition.

LARYNGEAL VERTIGO

(Laryngeal vertigo, Ictus Laryngis)

This is a condition in which vertigo or temporary unconsciousness occurs due to spasm of the true vocal folds with glottic closure and seen in males (men and boys).

The violent cough empty the air completely from lungs.

The excessive inspiratory effort holds and tends to maintain the the vocal folds in the adducted position, after complete emptying of air from the lungs and resulting into a negative pressure which leads to pulmonary venous pooling. This causes decreased cardiac output and cerebral ischemia leading to change in consciousness.

Causes

- Chronic bronchitis
- Chronic pulmonary disease
- Emotional instability
- Stress.

Precipatating factors

- Impacted wax
- Chronic cough with chest disease.

Clinical features

Symptoms

- Burning sensation in the larynx
- Paroxysms of cough
- Dizziness
- Light headedness
- Loss of consciousness
- Laryngeal spasm – Inspiratory stridor.

Signs

- Pale patient
- Cyanosis.

It should be differentiated from Epilepsy by the absence of convulsions.

Management

- Treat the underlying cough
- Psychotherapy .

DYSPHONIA PLICAE VENTRICULARIS

(False vocal fold phonation)

The use of supraglottic structures to produce sound results in a harsh, low pitched, strangled voice. (Mainly false vocal cords involved). The false vocal folds act as true vocal cords, as patient attempts speech.

It is mainly due to poor function of true vocal folds, like weakness or scarring of the true vocal folds or compensation of the false cords as true vocal cords can not approximate due to large polyps or nodules.

Patients prone for false vocal fold phonation

- Prolonged endotracheal intubated patients. (Injury to Cricoarytenoid joint)
- Radiation therapy undergoing cancer patients.
- Cancer patients who has undergone partial cordectomy. (Mucosal loss)
- Atrophied, bowed true vocal folds.
- Involvement of the true vocal folds by benign or malignant neoplasms.

Clinical features

- low pitched, harsh, monotonous strangled voice.
- Abnormal sounding voice .
- Voice fatigue with raw sensation in the throat.
- IDL shows that false cords hide the true cords while phonation and irregular vibration of false vocal folds.

Treatment - Voice therapy

PUBERPHONIA (Functional Falsetto voice of males, Mutational Falsetto)

This condition is seen in adolescent shy, insecure boys due to growth of larynx which causes drop in voice pitch. They present as high – pitched, feminine sounding (falsetto) voice. The elevation of larynx seen during phonation.

IDL reveals normal vocal cords.

It is confirmed eliciting chest register by firm downward pressing of the thyroid notch, while phonation.

Treatment – Supportive and intensive voice therapy.

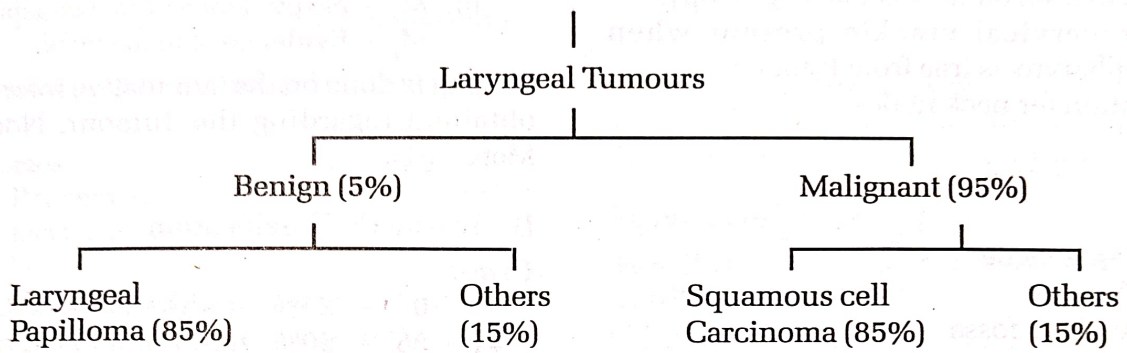
Psychotherapy.

CHAPTER 58

Tumours of the Larynx

CARCINOMA OF THE LARYNX

Laryngeal Tumours may be benign or malignant and the relative incidence is -



Therefore, Squamous cell Carcinomas form a vast majority of all laryngeal tumours.

Etiology

i. Age – Usually after the age of 40 years
ii. Sex – Common in males (M:F) = 8:1
iii. Predisposing factors
- Chronic vocal strain
- Smoking
- Alcohol
- Leucoplakia
- Papilloma
- Chronic granulomatous diseases.

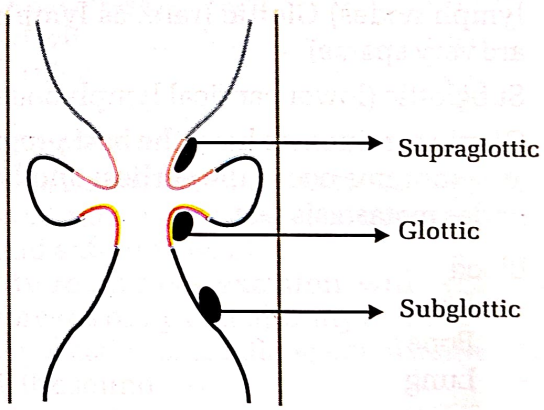
Gross Pathology

Sites:

i. Supraglottis (35%)
ii. Glottis (60%)
iii. Subglottis (5%)
iv. May spread to the Pyriform fossa.

Types

i. Malignant ulcer
ii. Mass
iii. Warty growth



Tumour sites

Microscopic Pathology

i. Squamous cell Carcinoma
ii. Verrucous carcinoma
iii. Ca-in-Situ
iv. Adeno carcinoma
v. Anaplastic carcinoma
vi. Sarcoma.

Symptoms

i. Hoarseness of voice
ii. Dry irritating cough and foreign body sensation
iii. Throat pain, (earache referred)

iv. Difficulty in swallowing (pyriform fossa involvement)
v. Hemoptysis
vi. Respiratory distress (pyriform fossa involvement)
vii. Neck mass (lymph node metastasis)

Signs

i. Growth seen on indirect laryngoscopy.
ii. Thyrocervical crackle present when cricopharynx is free from tumour.
iii. Palpation for neck nodes.

Spread:

1. Local
   - Base tongue
   - Thyroid
   - Pyriform fossa
2. Lymphatic - Supraglottic (upper cervical lymph nodes) Glottic (rare, as lymphatics are very sparse)

   Subglottic (lower cervical lymph nodes)

   Glottic carcinomas have the best prognosis as symptoms occur the earliest and lymph nodes metastasis last.
3. Blood
   - Liver
   - Bone
   - Lung
4. Transluminal - Especially when a tracheostomy has been done, spread occurs to the tracheostomy site.

Staging:

I. TNM Classification

The tumour (T) remains the same for any of the three anatomical areas of the larynx viz. supraglottis, glottis and subglottis.

i. T_0 - No evidence of primary tumour
   T_1 - Tumour confined to one anatomical region
   T_2 - Tumour extension to adjacent anatomical region
   T_3 - Transglottic tumour or extension/ fixation of one or both cords

ii. N_0 - No evidence of Lymph node involvement
   N_1 - Lymph node less than 3 cm in size on same side
   N_2 - Lymph node 3 cm to 6 cm in size on same side
   N_3 - Lymph node larger than 6 cm. on same side or contralateral nodes.

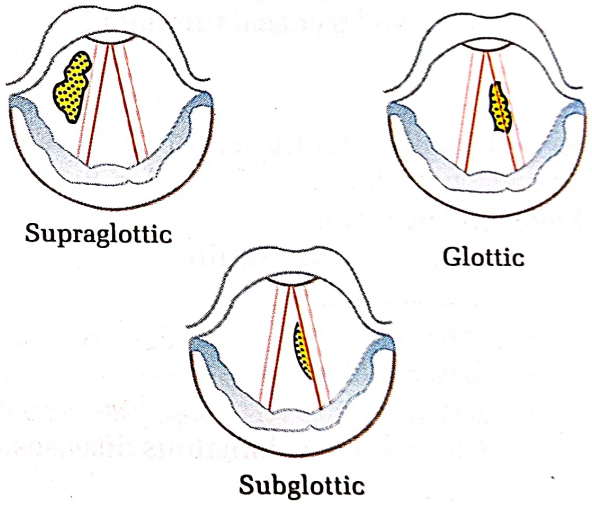
iii. M_0 - No evidence of metastasis.
   M_1 - Evidence of metastasis.

Staging is done on the cumulative information obtained regarding the Tumour, Node and Metastasis.

II. Broader's Classification

Grade:

i. 0 - 25% undifferentiated cells
ii. 26 - 50% undifferentiated cells
iii. 51 - 75% undifferentiated cells
iv. 76 - 100% undifferentiated cells.



Malignancy of the Larynx

Classification

Greater the grade of a tumour, worse is the prognosis.

Management:

1. Surgery – Laryngectomy.

Advantages

i. Response is good even for larger tumours
ii. Full treatment at one sitting (as patient compliance is usually not good).

Disadvantages

i. Loss of speech
ii. Anosmia, as air stream bypasses upper air passages.
iii Chances of foreign body aspiration are high as protective laryngeal reflexes are lost.
vi. Patient cannot strain during-lifting of heavy weights, coughing, defecation, mountaineering etc.
vii. Patient has a permanent tracheostoma.

2. **Radiotherapy** – 5000 rads over 5 weeks period.

Advantages

i. Preservation of speech and other laryngeal functions.
ii. Decreases size of the tumour
iii. Controls peripheral growth of the tumour
iv. Useful when patient is unwilling or unfit for Surgery

Disadvantages

i. Not useful for large tumours
ii. Chances of flap necrosis are high
iii. Wound break down and fistula formation may occur if followed by Surgery.
iv. Chances of initiating malignancy at another normal site (second primary).

3. **Chemotherapy**

Disadvantages

i. Very expensive
ii. Results are short lived. Recurrence of the tumour occurs within months.
iii. Side effects of various chemotherapeutic agents require constant monitoring of cardiac, renal, pulmonary and hepatic functions.

Treatment:

Stages
I - Radiotherapy
II - Radiotherapy or Surgery
III - Surgery
IV - Combined modality of treatment.

MULTIPLE JUVENILE LARYNGEAL PAPILLOMA

The name in itself is self-explanatory regarding the type of tumour, however not always confined to the larynx.

Etiology

i. Age – chiefly between the age of 5 and 15
ii. Sex – equal in both sexes
iii. Regression – occurs always before puberty
iv. Etiological factors
- viral infection
- hormonal imbalance
- amino-acid deficiency
- genetic.

Gross Pathology

i. Multiple warty growths
ii. Involving usually the inlet of larynx.
iii. Neighbouring mucosa is intact.

Microscopic Pathology

Papillary processes are seen which are surrounded by whorls of desquamated keratin.

Symptoms

i. Hoarseness of voice
ii. Cough
iii. Stridor.

Treatment

i. Medical treatment with Tetracyclines, systemic hormones and bovine wart vaccine and antiviral agents.
ii. Microsurgical excision with sharp cup forceps or by a diathermy suction.
iii. Application of caustic agents like silver nitrate
iv. Ultrasound
v. Cryosurgery
vi. LASER (treatment of choice).

Prognosis

Good invariably they disappear by puberty. However, tracheostomy is done without any hesitation as unfortunately these tumours show a tendency to recur with spread to almost any site in the larynx, pharynx or trachea. Sometimes they also reach the external tracheostomy orifice.

Juvenile Papilloma	Adult Papilloma
1. In children only	In adults
2. Multiple	Single
3. Tendency to recur	Does not recur
4. Disappears with puberty	Does not disappear
5. Never turns malignant (unless irradiated)	Malignant transformation may occur.

CHAPTER 59

Post-Laryngectomy Speech

A good speech rehabilitation is necessary for patients having undergone a laryngectomy, to prevent them from going into depression or a lowering of their morale.

Normal speech production involves 3 systems.

1. ***Respiration*** – provides the power for speech.
2. ***Phonation*** – occuring by vibration of the vocal cords is useful to generate a train of impulses of sound.
3. ***Articulation*** – modulates the amplitude of sound thus imparting speech its syllabic structure. Articulatory movements give rise to 2 basic types of sound.
   i. *Vowels* – in which the vocal tract is relatively open above the glottis.
   ii. *Consonants* – in which it is constricted or completely obstructed for some time.

Methods:

I. Oesophageal Speech

Mechanism – The patient is taught to gulp air and then belch out the air. This produces sounds by vibration at the cricopharygeal level and this sound is imparted quality by the palate, tongue, lips, nose etc.

Advantages

Simple procedure and involves no expense.

Disadvantages

1. Procedure has to be taught to the patient.
2. Patient is able to speak few words per phrase due to limited quantity of air gulped.
3. The speech is gruff, so unsuited for females.
4. Motivation of the patient is required.

II. Tracheo – Oesophageal Fistula

Mechanism – These methods are based on the principle that air is shunted from the pulmonary system to the oesophagus, which produces vibration of the local tissues and thus a tone audible for speech can thereby be generated.

Advantages

1. Here the air stream is produced by pulmonary exhalation as in normal speech, rather than the air gulping method of oesophageal speech. The speech is thus better controlled and provides more words per phrase.
2. Learnt immediately, as does not require prolonged training with speech therapist.

Disadvantages

1. Involves another surgery
2. Aspiration
3. Stenosis.

III. Electronic Larynx

Mechanism – An electronically produced vibrating sound source is present in this device and the patient uses the normal articulatory mechanisms to speak with the basic electronic sound. The loudness and pitch of the sound can be controlled by an external adjustable ring.

Advantages

1. Speech quality, duration and acceptability is much better
2. Procedure does not require to be taught
3. Does not prolong hospitalisation.

Disadvantages

1. Prominent external device
2. Expensive.

CHAPTER 60

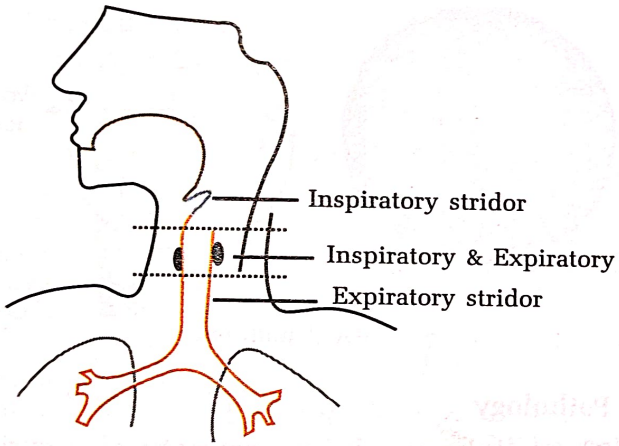
Miscellaneous Diseases of the Larynx

STRIDOR

Stridor is noise made by the obstruction to passage of air into or out of the lower respiratory tract.

Types :

1. Inspiratory phase – obstruction at laryngeal level.
2. Expiratory phase – obstruction at bronchial level.
3. Both phases – obstruction at tracheal level.



Types and site of origin

Etiology – Causes:

1. *Congenital*
   i. laryngomalacia
   ii. bifid epiglottis
   iii. laryngeal web
2. *Inflammatory*
   i. acute laryngitis
   ii. acute laryngotracheobronchitis
   iii. diphtheria
   iv. measles
   v. whooping cough
   vi. tuberculosis
   vii. tetanus.
2. Traumatic
   i. birth injury
   ii. intubation
3. Neurological
   i. neonatal tetany
   ii. tetany (laryngismus stridulus)
   iii. recurrent laryngeal nerve paralysis

II. Acquired

i. acute epiglottitis
ii. acute laryngitis
iii. acute laryngotracheobronchitis
iv. diphtheria
v. foreign body.

Diagnosis of cause:

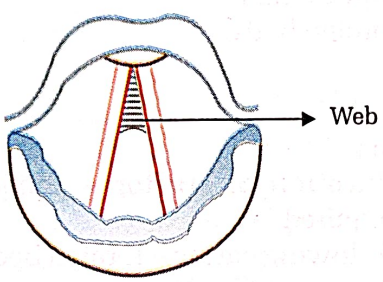
1. **History**
   i. Duration of stridor – congenital or acquired
   ii. Following eating – foreign body
   iii. Feeding difficulties, cough or choking
   iv. Generalised illness – measles, tuberculosis
   v. Sore throat – diphtheria
   vi. Hoarseness in the past – papilloma.
2. **Examination**
   i. For respiratory distress
   ii. Throat examination
   iii. Respiratory system examination
   iv. X-ray neck and chest
   v. Laryngeal examination.

Management

1. A patient in stridor is hospitalized
2. Complete clinical examination
3. Oxygen
4. Steroids and antibiotics
5. Tracheostomy or intubation if the obstruction is severe
6. Treatment of cause.

CONGENITAL CONDITIONS OF LARYNX

1. ***Laryngomalacia*** – is the commonest disorder. The laryngeal cartilages show excessive flaccidity resulting in indrawing of the laryngeal inlet during inspiration. On direct laryngoscopy the inlet reveals a curled or omega shaped epiglottis and the symptoms usually disappear by 2 years .
2. ***Subglottic stenosis*** – occurs due to a thickening of the cricoid cartilage below the vocal cords. Most of them improve with age as the larynx becomes bigger.
3. ***Laryngeal web*** – generally occurs at the anterior part of the vocal cords. It cause biphasic stridor with hoarse cry. The web is cut using the laser beam.



Laryngeal web

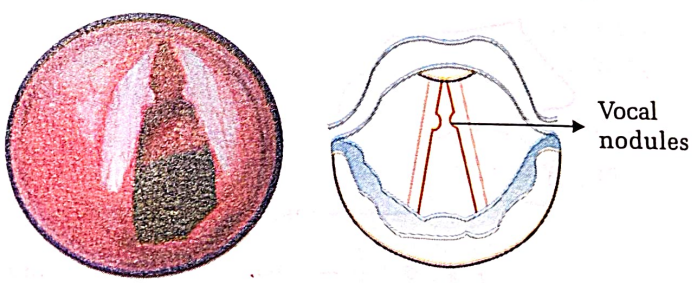
4. ***Vocal cord paralysis*** – is due to birth trauma or specifically following forceps delivery affecting the recurrent laryngeal nerve.
5. ***Laryngocoele.***
6. ***Sub glottic haemangioma*** – associated with other cutaneous hemangiomas in children. Manifestation takes place between 3 to 6 months as the hemangiomas grow in size.A mediastinal extension is also to be ruled out in these children. Steroids and laser ablation is the treatment, tracheostomy may be required at times.
7. ***Laryngo-oesophageal cleft*** – is due to failure of fusion of cricoid lamina. Aspiration and pneumonia are the complications.During feeds the child starts coughing or gets choking episodes.
8. ***Laryngeal Cyst*** – is a mucus filled sac which narrows the laryngeal inlet, causing stridor. Usually situated in the aryepiglottic folds and treated by endolaryngeal marsupialisation or by Laser ablation.

VOCAL NODULES (Singer's nodes)

A condition which occurs in persons who use their voice excessively, with straining or by faulty production.

Etiology

1. ***Age*** – seen after puberty.
2. ***Sex*** – occurs more often in females.
3. ***Occupation*** – seen in singers, teachers, hawkers, preachers etc.



Vocal nodules

Pathology

The nodules are always symmetrical, occuring on both cords at the junction of the anterior with the middle one-third. In established cases the overlying epithelium is seen to have undergone a degree of hyperplastic thickening. However, the nodule remains entirely superficial and does not hinder with the rhythmical flowing movement of the epithelium during phonation.

Clinical features

1. Increasing hoarseness of voice
2. Vocal fatigue
3. Inability to sing high notes softly, is diagnostic
4. Indirect laryngoscopy reveals symmetrical small nodules at the anterior and middle-third junction of the vocal cords.

Treatment

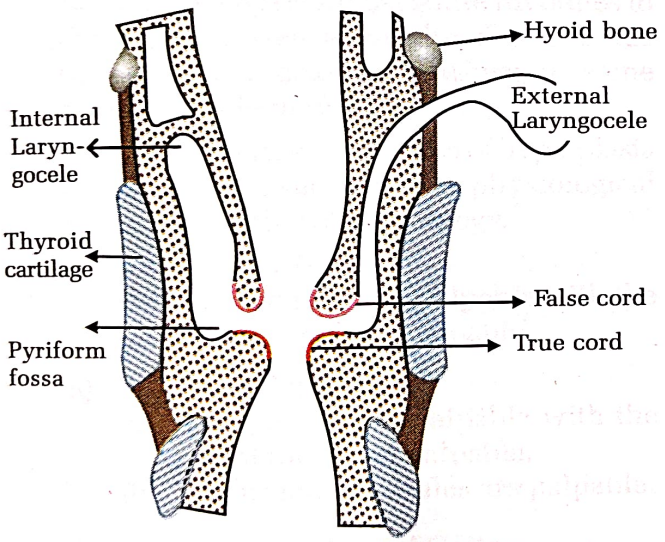
1. *Voice rest* – is essential and may cure recent and small nodules.
2. *Septic focus* – in tonsils, sinuses, teeth etc are treated.
3. *Excision* – of the nodules by (MLS) microlaryngoscopy.
4. *Speech therapy* – to prevent a recurrence as it helps to re-eduacate regarding voice production.

LARYNGOCELE

Laryngocele is an air filled sac, neither a cyst nor a tumour. It may become a pseudocyst after its neck is obliterated by inflammatory changes.

Types :

1. External
2. Internal
3. Mixed



Laryngocele

Etiology

1. Age – seen in adults
2. Sex – Commoner in males
3. Occupation – players of wind instruments, glass blowers etc.

Clinical features

1. Neck swelling
2. Voice changes
3. Acutely inflammed cyst.

Diagnosis

1. A bulge is felt on coughing or Valsalva's manoeuvre.
2. Completely disappears on pressure.
3. X-ray during Valsalva (expiration).

Management

1. Treatment required only if troublesome symptoms are present.
2. Excision via an external approach.

EDEMA OF THE LARYNX

It is not a disease as such because edema of the larynx may be the most conspicuous feature of the inflammatory reaction in a wide variety of infective and non-infective diseases of the larynx.

Etiology

1. **Inflammatory**
   - i. Acute laryngitis
   - ii. Chronic laryngitis
   - iii. Typhoid
   - iv. Laryngotracheobronchitis
   - v. Tuberculous laryngitis
   - vi. Mumps
   - vii. Perichondritis
2. **Trauma**
   - i. Strangulation and cut throat injury
   - ii. Intubation
   - iii. Inhalation of fumes
   - iv. Irradiation
3. **Malignancy**
   - i. Base tongue
   - ii. Pyriform fossa
   - iii. Larynx
4. **Angioneurotic edema**
5. **General causes**
   - i. Cardiac
   - ii. Hepatic
   - iii. Renal
   - iv. Obstruction of superior vena cava

Pathology

'Edema glottis' was the term previously used to describe laryngeal obstruction caused by laryngeal edema. In fact, the glottis is not involved in these cases, edema occurs in the submucosa of the supraglottic and or subglottic tissues.

Clinical features

Due to its location, it interferes with breathing rather than voice.

Treatment

1. Cause is treated
2. Steroids
3. Tracheostomy may be required.

CHAPTER 61

Thyroid

1. Physiological Goitre:

It is a butterfly shaped swelling in the midline of the neck and extending on either side of the trachea and is firm in consistency.

Precipitating factors:

(a) *Milestones in female* life such as puberty, pregnancy, bad obstetric history.

(b) *Stress factors* such as debilitating disease or mental stress. These factors increase the demand for thyroxine, producing a relative deficiency state.

2) Nodular Goitre:

Normally a physiological goitre involutes to the normal size after the demand for thyroxine has ceased. However in some cases a nodule may form.

A nodule is defined as an area of hyperplasia and involution secondary to physiological abberation in thyroid pathology.

i) **Uninodular Goitre :**
Only a single nodule palpable with the rest of the gland *not* palpable.

ii) **Multinodular Goitre:**
(a) A single nodule palpable with the rest of the gland palpable.
(b) Two or more nodules are palpable.

3. Parenchymatous/Colloid Goitre:

Bilateral swelling in the midline of the neck extending on either side of the trachea with a smooth surface and soft consistency.

When the demand for thyroxine ceases a physiological goitre involutes. If the whole gland hyperinvolutes forming enlarged follicles filled with colloid, it forms a colloid goitre.

4. Carcinoma of the Thyroid:

Histopathological types
a) Papillary
b) Follicular
c) Medullary
d) Anaplastic

Clinical types

i) Clinically occult.
ii) Clinically suspicious - when the size of the gland increases rapidly with pain and symptoms of dysphagia, hoarseness or dyspnoea.
iii) Clinically evident - when a hard, nodular or plaque like thyroid can be palpated.

5. Thyroiditis:

i) Hashimoto's thyroiditis (autoimmune).
ii) De'Quervans thyroiditis (viral).
iii) Riedle's thyroiditis (pyogenic).

HISTORY

1. General particulars

- Age
- Occupation
- If from endemic area (Sub-Himalayan Plateau or in Maharashtra foot hills of Satpuda, hule).

2. **Chief complaints** and origin, duration and progress to be recorded.

3. **Rule out lymph node disease of neck** i.e. Koch's/Pyogenic.

4. **Enquiry for the precipitating factors:**
a) Milestones in female i.e. puberty, pregnancy, bad obstetric history, menopause.

b) Stress i.e. debilitating illness, mental stress.
c) Ingestion of goitrogens such as cabbage, spinach, contaminated fish etc.
e) Familial predisposition.
f) Endemicity.
g) Metastasis to lung, liver, brain or bone.
h) Tuberculosis.
i) Previous treatment taken.

5. Assess complications

a) Complications due to pressure
i) Hoarseness of voice (recurrent laryngeal nerve).
ii) Dysphagia (oesophagus)
iii) Dyspnoea (trachea)
iv) Syncope (Internal carotid artery)
v) Horner's syndrome (cervical sympathetic chain)

b) Complications of the swelling:
i) Presents as sudden increase in size of the swelling with pain.
ii) Malignancy:
Rapid increase in the size with hoarseness,
H/o loss of weight and appetite H/o haemoptysis, bone pains, jaundice or convulsions.
iii) Thyroid functional status:
a. **History of Hyperthyroidism:**
- **Metabolic:**
H/o excessive appetite with loss of weight.
- **CNS effects:**
Insomnia
Irritability
Anxiety
Crying spells
Easy fatiguability
Fine tremors
Exaggerated reflexes

Quadriceps sign : Feeling of give way of the knees while climbing down stairs.

- **CVS effects:**
Chest pain
Palpitations
Syncope
- **GI effects:**
Abdominal colic
Abdominal hurry
- **GU effects:**
(Urinary system is not affected)
Menorrhagia
Polymenorrhea

b. H/o Hypothyroidism:
- **Metabolic:**
Decreased appetite and abnormal weight gain
Intolerance to cold
Malaise
Lethargy
Somnolence
Sluggish reflexes
- **CVS effects:**
Chest pain
Palpitations
Syncope
- **GI effects:**
Constipation
- **GU effects:**
Oligomenorrhea
BOH

EXAMINATION OF THE SWELLING

1. General Examination:
- Pallor
- Icterus
- Lymphadenopathy

Virchow's Lt. supraclavicular node (if palpable)

Signs of Toxicity :

- **Fine tremors of the fingers/tongue**
These can be noticed by asking the patient to extend his elbow and fan out his fingers. A piece of paper is then placed over the fingers and movement of the paper should be looked for.
- **Eye signs of thyrotoxicosis.**

a) Von Graefe's lid-lag sign:
The upper eyelid lags behind the eyeball as the patient is asked to look downwards suddenly.

b) **Dalrymple's sign:**
Visibility of the sclera above the iris (indicates clinical exophthalmos).

c) **Joffroy's sign:**
Absence of wrinkling of the forehead, when the patient looks upwards with the head fixed.

d) **Moebius's sign:**
Lack of convergence of the eyeballs.

Sleeping Pulse rate:

Normal or hyperdynamic water hammer pulse.
i) < 90 mild thyrotoxicosis.
ii) 90-110 moderate thyrotoxicosis.
iii) 100 severe thyrotoxicosis.

Blood Pressure : High pulse pressure is seen in thyrotoxicosis.

2. Local Inspection:

i) A swelling
ii) A thyroid swelling
iii) Functional.

This is carried out with the patient in the ***Sitting position with slight passive extension of the neck (Pizillo's Method).***

Look for :
- Size
- Shape
- Situation: usually a midline swelling extending on either side of the trachea.
- Extent:

i) Laterally the relation of the lateral border to the sternocleidomastoid.
ii) Superior extension should be mentioned.
iii) Lower border seen clearly move upwards with deglutition or
Extending upto suprasternal notch.
- Skin over the swelling:
- Signs of inflammation
- Scars
- Sinuses
- Dilated veins

Movement on deglutition:

Ask the patient to swallow and look for upward movement of the swelling. The thyroid is attached to thyroid cartilage by the pretracheal fascia which clothes the gland. The thyroid cartilage moves up with deglutition pulling the gland along with it. Also notice if the inferior border of the gland can be appreciated. (Movement upwards after protrusion of the tongue - should be mentioned only if swelling is single and in midline).

Platysma sign:

Due to prominence of the platysma due to malignant infiltration.

Pemberton's sign:

Congestion of face on raising of hands.

3. Palpation:

The examiner stands behind the patient with the neck of the patient passively flexed. Active flexion against resistance will contract the sternocleidomastoid.

Look for - local warmth and tenderness over the swelling.
- Signs of inspection are to be confirmed.
- Surface of the swelling. To palpate the posteromedial surface of the gland use **Lahey's manouvre.**
- Push the gland to one side by the fingers of one hand and palpate the posteromedial surface with the thumb of the other hand.
- **Crile's method:** Palpate a lobe only with the thumb. Ask the patient to deglutate and feel for small nodules on the surface.
- Consistency of the swelling. This can be soft, firm, cystic or hard.

Relation to the surrounding structures:

(a) Sternocleidomastoid muscle:
Palpate the lateral border of the swelling and note whether it extends deep to the muscle. To contract the right Sternocleidomastoid the patient is asked to look to the left and upwards against resistance and vice versa.

b) Inferior border of the gland:
Palpated before and after deglutition. In case of a retrosternal goitre the inferior border is not palpable even after deglutition.

c) **Carotid pulsations :**
In normal individuals carotid pulsations are palpable against the tubercle of the transverse process of C_6 vertebra (Chassagnaec tubercle).
In case of a goitre the carotids are to be palpated against transverse process of C_2 vertebra i.e. distal to the gland superiorly.

Berry's sign : is obliteration of carotid artery pulsations (in malignancy).

d) **Trachea:**
The position of the trachea should be confirmed by palpating the trachea in the suprasternal notch. In case of a large goitre where the trachea may not be palpable, the Kocher's test may be of help. Pressure over the goitre from the lateral to the medial side induces a gag from the patient. This is the position of the trachea. (done only in a large goitre enlarged on either side of trachea).

e) **Superior Vena Cava (SVC):**
In case of a retrosternal goitre, ask the patient to hold the breath in inspiration and raise his arms above the shoulders. Dilatation of veins over the face and neck indicates pressure over the SVC (Pemberton's Sign).

f) **Loss of ciliospinal reflex** Horner's syndrome indicates pressure over the cervical sympathetic chain.

g) **Lymph nodes :**
Examination of cervical nodes should be carried out in detail as palpable, hard, fixed, nontender nodes may be the only indicator of an occult carcinoma of the thyroid. (It must be remembered that metastatic lymph nodes are *always hard* in thyroid carcinoma).

4. **Percussion:**
A dull note over the sternum with heavy percussion is suggestive of a retrosternal goitre.

5. **Auscultation:**
A bruit may be appreciated at the superior pole of the gland.

6. **Systemic Examination:**
   a) **CVS:**
      - S_1 and S_2
      - Murmur (of hyperdynamic circulation)
      - Arrythmias
      - Water hammer pulse
   b) **CNS:**
      - Cranial nerves
      - muscle tone and power
      - reflexes
   c) **Respiratory:**
      - Position of trachea
   d) **G. I. T.** (if contributory)

7. **Investigations:**
   i) **Sleeping pulse rate:**
   The pulse rate is taken 2 hours before the scheduled time of awakening. This allays the effect of anxiety on the pulse rate.

   ii) **Indirect laryngoscopy (IDL)**
   This is done to determine the state of the vocal cords prior to surgery.

   iii) **X-ray neck** (AP and lateral) to note any tracheal deviation or compression which would make intubation difficult.

   iv) **X-ray chest:**
      a) Retrosternal extension
      b) Metastasis
      c) Anaesthesia fitness.

   v) **ECG:**
      a) Tachycardia
      b) Arrythmias
      c) Ischaemic changes.

   vi) **Radio Isotope scans:**
   Using Radiolabelled compounds to demonstrate the functional status of the gland. The nodules may be hot or cold.

   vii) **Thyroid hormone assay** T3, T4, TSH levels.

   viii) **USG -**
      a) Site, size and number of nodules
      b) Solid/cystic
      c) Lymph nodes
      d) Guided FNAC

ix) **FNAC:**
Provides histopathological diagnosis. However diagnosis of follicular carcinomas is not reliable on FNAC.

Diagnosis:

a) Toxic / Non - toxic
b) Physiological / Colloid / Nodular
c) Suspicion of malignancy
d) Retrosternal extension
e) Compression of trachea or oesophagus

Modalities of treatment are:

1. MEDICAL

a) **Replacement therapy** with 0.1 mg of thyroxine per day in case of a physiological goitre till demands are met as indicated by T3, T4, TSH levels.

b) **Antithyroid drugs:**
i) **Propylthiouracil** 50-100 mg TDS followed by 20-30 mg TDS for maintenance.
ii) **Carbimazole** 5-10 mg TDS initially, maintenance dose 2.5-10 mg daily in 1 to 3 divided doses.

c) **Pre-operative preparation** for a toxic goitre:
i) to make the gland euthyroid.
ii) to make the gland firm and less vascular to help dissection.

Drugs used:

1) **Carbimazole** 5-10 mg TDS initially to achieve a euthyroid state followed by maintenance dose of 2.5 - 10 mg TDS.
2) **Lugol iodine** - This is given 10 days prior to surgery in a dose of 5-15 drops per day discontinued 48 hours prior to surgery.
3) **Thyroxine** 0.1 mg per day may be added to prevent hypothyroid state.
4) **Propanalol** may be given to control the heart rate.

2. SURGERY

This is a definitive mode of therapy.

a) **Excision enucleation:**
Excision of a nodule with 1.5 cm of normal surrounding thyroid tissue.

b) **Hemithyroidectomy:**
Excision of one lobe with whole of the isthmus.

c) **Partial:**
Removal of the gland leaving behind enough thyroid tissue so as to fill the tracheo-oesophageal gutter, to protect the recurrent laryngeal nerve and parathyroids and to maintain metabolism.

d) **Subtotal thyroidectomy:**
Removal of as much gland as possible leaving behind 2 thin strips of tissue in the tracheo - oesophageal gutter so as to protect the recurrent laryngeal nerve and parathyroids to maintain metabolism.

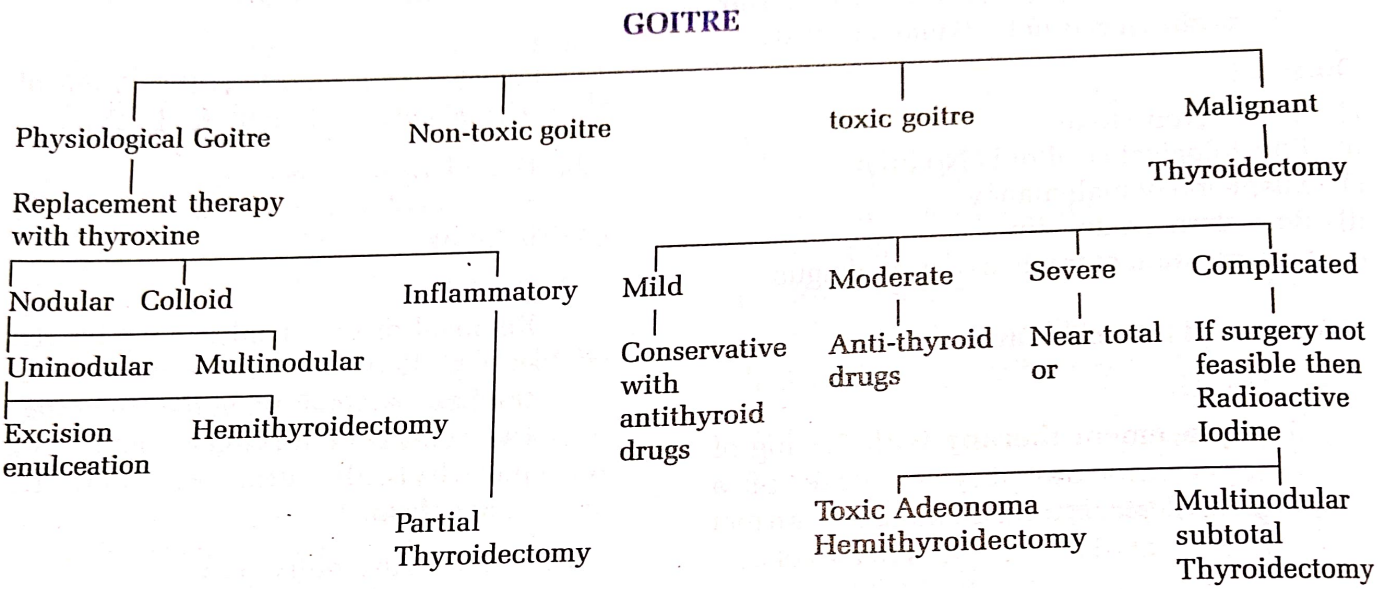
e) **Near Total or Total thyroidectomy:**
Removal of the whole of the thyroid gland leaving behind tissue enough to protect the recurrent laryngeal nerve and parathyroid glands.

3. RADIOACTIVE IODINE:

The isotope used as I_{131} which is concentrated by the thyroid gland.

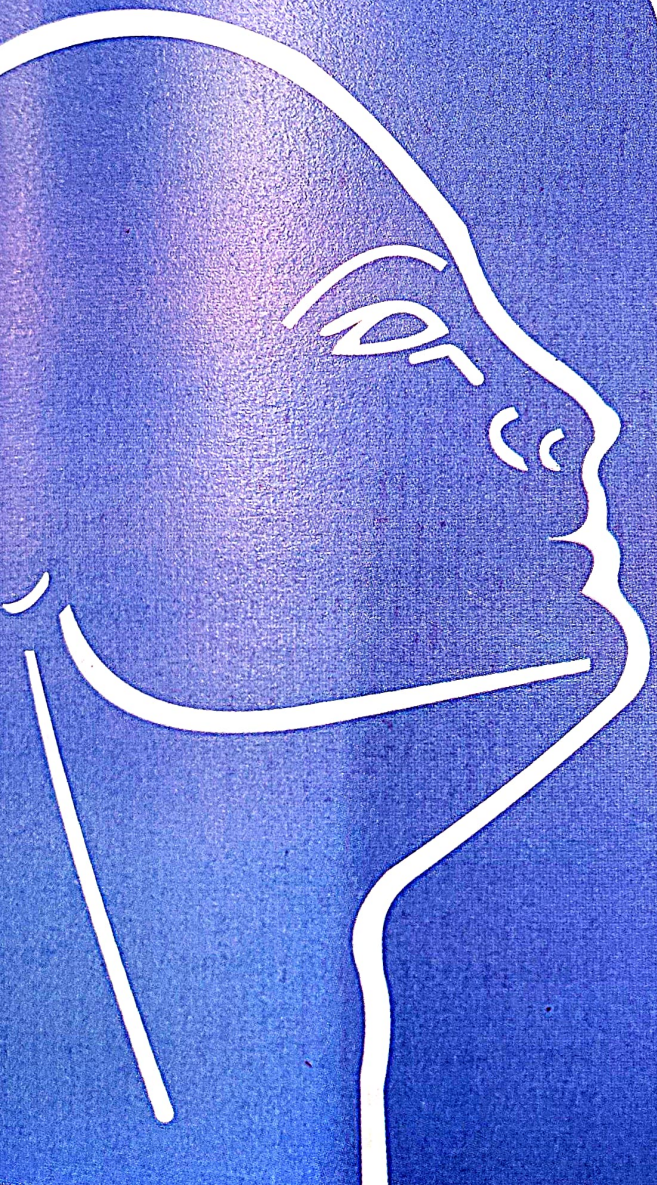
It emits Beta - rays which destroy the thyroid cells, The dose is individualised and the thyroid status is evaluated after 3 months of therapy. The dose may be repeated if required.

Treatment:


GOITRE
Physiological Goitre
Replacement therapy with thyroxine
Non-toxic goitre
Nodular
Colloid
Uninodular
Multinodular
Excision enulceation
Hemithyroidectomy
Inflammatory
Partial Thyroidectomy
toxic goitre
Mild
Conservative with antithyroid drugs
Moderate
Anti-thyroid drugs
Toxic Adeonoma Hemithyroidectomy
Severe
Near total or
Complicated
If surgery not feasible then Radioactive Iodine
Multinodular subtotal Thyroidectomy
Malignant
Thyroidectomy

SECTION - VII

TRACHEO-BRONCHIAL



CHAPTER 62
Bronchoscopy

It is an endoscopic direct visualisation of the Tracheo-Bronchial tree using a bronchoscope.

Indications:

I. Diagnostic

1. Unexplained
   - Chronic cough
   - Sputum production
   - Stridor
   - Hemoptysis
   - Wheeze
   - Dyspnoea
   - Collapse
2. Suspected Foreign body
3. Vocal cord palsy
4. Spread of cancer from Thyroid or Oesophagus
5. Sputum cytology
6. Tracheo-Oesophageal fistula

II. Therapeutic

1. Foreign body removal
2. Aspiration of secretions
3. Aspiration of lung abscess
4. Removal of Broncholith
5. Dilatation for Bronchial stenosis
6. Removal of benign tumours like papilloma
7. Lung lavage in Asthma
8. For difficult intubations.

Contraindications:

1. Generalised Medical disorders
2. Bleeding diathesis
3. Aortic aneurysm
4. Trismus
5. Cervical spondylosis or fracture of cervical spine
6. Recent massive hemoptysis of any cause.

Technique

1. Done under General Anaesthesia. The ventilation is maintained with a jet instrument called *Venturi*, supplying oxygen at a very high pressure, this works on the Bernoulli's principle. Maintaining ventilation adequately is very important here as the surgeon and anaesthesist share the same pathway (Trachea and Bronchus).
2. Patient lying supine in *Boyce's position*.
3. The scope is held in the right hand and guided with the left hand.
4. It is passed along the base of tongue and then the epiglottis is lifted with its tip. The scope is now turned by 90° so that it can pass through the anteroposterior axis of the cords and prevents damage to the cords.
5. The scope now enters the trachea, which is confirmed by :
   i. Blast of Air
   ii. Tracheal rings
   iii. Movements with respiration.
6. The scope is passed further downwards too visualise the carina for any widening.
7. The head is then flexed towards the left and scope passed into the right bronchus. Then the head is flexed towards the right to pass the scope into the left bronchus.
8. On removal of the scope, care is taken not to damage the vocal cords.

Complications

1. Anaesthesia
2. Damage to lip, teeth, tongue, arytenoids or vocal cords
3. Cardiac arrest
4. Sub-glottic edema
5. Tracheal tear (very rare).

CHAPTER 63

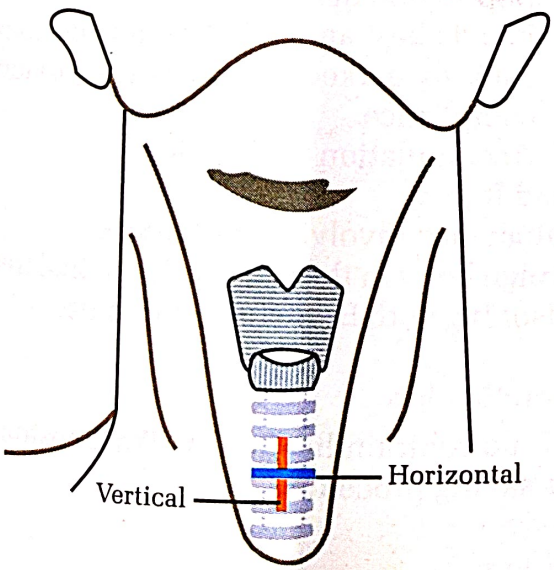
Tracheostomy

Definition – Opening made on the anterior wall of trachea to bypass the upper air passages and maintain ventilation between the outside and the trachea.

Classification:

A.

1. **High-Tracheostomy**
   Done above the isthmus of the thyroid gland. Usually at the 1st tracheal ring. Never performed as damage to cricoid cartilage occurs, which later leads to laryngeal stenosis.
2. **Mid-Tracheostomy**
   Done at the isthmus of the thyroid gland. Not performed as isthmus has to be clamped, cut and ligated. The 2nd or 3rd Tracheal ring is incised.
3. **Low-Tracheostomy**
   Done below the isthmus of the thyroid at the level of 4th Tracheal ring. This is the preferred site of tracheostomy.



Incision for Tracheostomy

B.

1. **Emergency Tracheostomy**
   If the patient is in stridor.
2. **Elective Tracheostomy**
   Done when respiratory distress is anticipated, prior to surgery.

C.

1. **Temporary Tracheostomy**
   Later, the tracheostomy tube is removed.
2. **Permanent Tracheostomy**
   Done after a laryngectomy, where tracheal mucosa is sutured to skin.

Functions of a Tracheostomy:

1. Reduction of dead space by one-third.
2. Direct access to the lower respiratory tract for suction and instillation of mucolytic agents.
3. Easy mode of intubating a patient when endotracheal intubation is not possible.
4. Relieves upper airway obstructions, by creating a bypass.
5. Allows patient to be kept on a Respirator for IPPR (Intermittent Positive Pressure Respiration).

Disadvantages:

1. Loss of speech, as air will bypass the larynx.
2. Loss of smell, as air does not pass through the nose.
3. Chances of foreign body aspiration are very high as Laryngeal protective mechanism is lost (epiglottis, false cords and true cords).
4. Inability to lift heavy weights or perform strenuous exercises as positive subglottic pressure cannot be built up.
5. Patient cannot swim.

Indications:

I. Obstructive

A. Instrinsic

1. Congenital	a. laryngeal web b. Laryngeal stenosis c. Laryngeal hemangioma
2. Traumatic	a. Cut throat injuries b. Vehicular accidents c. Strangulation
3. Inflammatory	a. Diphtheria b. Tuberculosis c. Fungal infections d. Scleroma e. Laryngotracheobronchitis
4. Neoplastic	a. Carcinoma of Larynx b. Laryngeal Papilloma
5. Paralytic	Bilateral abductor paralysis
6. Miscellaneous	a. Foreign body in the air passages b. Laryngeal edema c. Tracheomalacia d. Subglottic Stenosis.

B. Extrinsic

1. Congenital	- Choanal Atresia
2. Traumatic	a. Facio-maxillary injuries b. Chest Injuries
3. Inflammatory	a. Ludwig's Angina b. Bronchopneumonia
4. Neoplastic	a. Malignancy of Thyroid b. Lymphoma c. Malignancy of Oesophagus d. Bronchogenic Carcinoma e. Metastatic Neck nodes
5. Vascular	a. Hemangiomas b. Aneurysms.

II. Where Asphyxia is anticipated

1. Removal of Laryngeal papilloma
2. Laryngo-fissure surgery
3. Major Head-Neck surgery like hemi-mandiblectomy or glossectomy
4. Block dissection.

III. Respiratory Centre depression

1. Coma, which may be due to either Diabetic, Hepatic dysfunction, Uremia or Head Injury
2. Barbiturate poisoning
3. Base of Skull fractures
4. Intracranial space occupying lesions
5. Cerebro-vascular accidents
6. Raised Intracranial tension.

IV. Neural Pathology

1. Tetanus
2. Poliomyelitis
3. Cervical lesions
4. Polyneuritis like Guillain-Barre syndrome
5. Overdosage of muscle relaxants.

V. Therapeutic

1. For suction of excessive chest secretions
2. Putting the patient on a Respirator for more than 48 hrs
3. Instillation of mucolytic agents like Sodabicarb or Streptokinase for thinning secretions.

Common Indications:

1. Tetanus. Tracheostomy is life saving since there is :
   i. Laryngeal Spasm.
   ii. Depression of respiratory centre.
   iii. Fixed chest and opisthotonus position leads to marked decrease in respiratory compliance.
   iv. Accumulation of secretions.
2. Head Injury
3. Malignancy involving the Larynx
4. Foreign body in the tracheo-bronchial tree
5. Poisoning with Bronchopneumonia.

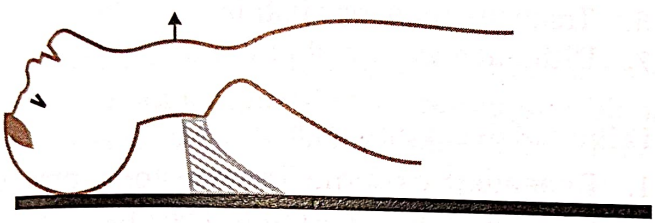
Contraindication

There is no contraindication, as Tracheostomy is a life saving procedure.

Technique:

1. Consent for Tracheostomy. No premedication is given.

2. The patient is placed supine with pillow kept below the shoulder so the trachea becomes prominent in the hyperextension position.
3. Usually done under local anaesthesia, 2% lignocaine is injected from the cricoid cartilage to the suprasternal notch along the midline.
4. A midline vertical incision is made below the cricoid cartilage upto 2 finger spaces above the suprasternal notch. A horizontal incision is avoided as can cause damage to neighbouring structures, however only has a cosmetic advantage as it is taken along the neck skin crease.



Position of hyperextension

5. Layers encountered from outside are
   i. Skin.
   ii. Platysma.
   iii. Investing layer of deep cervical fascia
   iv. Strap muscles.
   v. Pretracheal fascia.
6. The Thyroid isthmus is retracted and held upwards with a hook to create more space, to fix the trachea and to make the trachea superficial.
7. Trachea is identified by
   i. The cartilagenous white rings
   ii. A needle-syringe is put into the Trachea and air bubbles seen on aspiration. Then 2-3 drops of local anaesthetic are introduced into the tracheal lumen. This not only prevents a vasovagal attack but also suppresses the cough reflex.
8. Incision on the Trachea is made with a stab knife in the form of a T, X, Y or sometimes even a flap is removed.
9. Dialatation of the Trachea opening is done with a Tracheal dilator.
10. Tracheostomy tube is introduced along the curve of the Trachea.
11. The pilot is withdrawn immediately.
12. The air blast is checked with a thread and chest ausculated for air entry.
13. The tube is tied with a tape to the flanges on either side and around the neck. This keeps the tube in place.

Post Tracheostomy Orders:

1. An additional set of a tracheostomy tube is kept beside the patient.
2. Suction is performed every half hourly.
3. With a metal tube, the inner tube is removed, cleaned and reinserted 4 hourly.
4. For a Portex tube, the cuff is deflated 2 hrly for a period of 10 min to prevent pressure necrosis of tracheal cartilage.
5. Humidification is necessary as the air blast bypasses the upper airway. This is achieved by keeping a moist gauze piece over the tube opening.
6. Chest Physiotherapy is given for clearance of accumulated secretions.
7. Mucolytic agents are used for thinning out secretions thus helping in their removal.
8. Expectorants.
9. A bell is kept next to the patient, which can be used in case of respiratory distress.
10. Oxygen is given SOS.
11. Any change of Tracheostomy tube is done only after 48 hrs, when a tract has formed. If done earlier, it can result in spontaneous closure of the created opening.

Complications:

Anaesthesia

i. Anaphylaxis
ii. Cardiac Arrest
iii. Hypotension.

A) Immediate

1. Vasovagal attack
2. Hemorrhage from
   i. Isthmus of thyroid
   ii. Anterior jugular vein
   iii. Inferior thyroid vein
   iv. Thymus in infants
3. Damage to pleura in infants
4. Damage to oesophagus
5. Cricoid cartilage injury
6. Air embolism
7. Recurrent laryngeal nerve damage
8. Post tracheostomy apnoea.

B) Delayed

1. *Bleeding* due to erosion by the tip of the metal tube.
2. *Excessive crust formation* leading to obstruction of the tube.
3. *Surgical Emphysema* caused by closing the neck wound too lightly.
4. *Pneumomediastinum* may occur following surgical emphysema as inspiratory efforts may suck the air into the mediastinum from the neck. The air bubbles then compress the large veins and cause cardiac failure.
5. *Pneumothorax occurs* in infants due to the pleura being opened during surgery on account of its position in the neck.
6. *Slipping of the tube.*
7. *Dysphagia* due to
   i. Inability to create a positive pressure during deglutition.
   ii. Cuffed tube may press upon the oesophagus.
   iii. If a tracheal flap is sutured to the skin, it prevents the normal upward movement of the larynx during deglutition.

C) Late

1. Tracheitis
2. Tracheal stenosis due to a high tracheostomy
3. Tracheomalacia due to infection and necrosis of cartilage
4. Tracheo-oesophageal fistula
5. Bronchopneumonia
6. Tracheo-cutaneous fistula
7. Difficult decannulation.

Failure of decannulation

1. Dependence on the Tracheostomy tube is seen commonly in a child. The tube reduces the dead space of airway and therefore breathing is without much resistance.
2. The pathology for which Tracheostomy has been performed has not been fully treated.
3. Inadequate air passage due to Tracheomalacia or Tracheal stenosis following tracheostomy.
4. Psychological dependence on the Tracheostomy.

CHAPTER 64

Laser

LASER is Light Amplification by Stimulated Emission of Radiation; acts as a precise, bloodless, light scalpel or can be used for photo-coagulation. The clinical applications of laser depend upon their wavelength and the absorptive powers of the tissues where they are used.

Types

1. ***Carbon-dioxide*** Laser-wave length 10600nm, very effective to vapourise and give a bloodless field.
2. ***Argon Laser***-wave length 488-514nm, used for photocoagulation of retina and blood vessels. It has been used for stapedectomy, middle ear surgery and haemangiomas.
3. **KTP-532** ***Laser*** (Potassium titanyl phosphate) -wavelength 532 nm, used for micro surgery of the ear.
4. ***Nd-Yag laser*** (Neodymium / Yttrium Aluminium Garnet)-wave length 1060nm, can be transmitted by flexible endoscopes; also have coagulative properties. Used extensively for malignancy in ENT and tracheal or endo-bronchial tumours.

Laser-tissue interactions

When laser light strikes biologic tissue, light energy is converted to heat energy. The greater the laser energy absorbed, greater is the potential for hemostasis which takes place by the heat induced shrinkage of collagen component of the vessel wall.

Function of laser beam

1. Vapourise
2. Cut and
3. Coagulate the tissues.

Photomechanical and photochemical changes may also occur.

Indications:

A) Ear

i. Stapedectomy
ii. Myringotomy
iii. Acoustic neuroma
iv. Aberrent vessels in the middle ear

B) Nose

i. Membranous choanal atresia
ii. Intranasal adhesions
iii. Rhinophyma
iv. Papilloma
v. Angioma

C) Oral Cavity

i. Leukoplakia
ii. Early malignant tumours
iii. Submucous fibrosis

D) Larynx

i. Webs
ii. Cysts
iv. Vocal nodules
v. Polyps
vi. Juvenile papilloma
vii. Early malignant tumours
viii. Bilateral vocal cord palsy.

E) Trachea - Benign tumours
Recurrent papillomas
Granulation tissues.

Complication – with respect to

1. Anaesthesia

i. Ignition of endotracheal tube
ii. Explosion

2. Surgeon

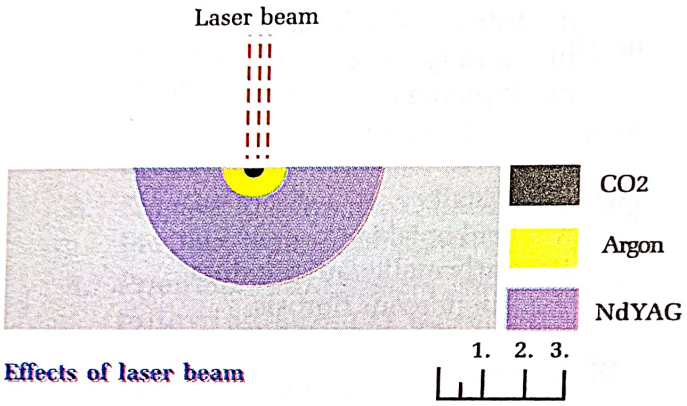
i. Retinal damage
ii. Burns

3. Patient

i. Burns
ii. Cord palsy due to thermic effect
iii. Bleeding, if vessel is larger than 0.5 mm in diameter.
v. Vocal cord damage.

Advantages

1. Achieves hemostasis for vessels less than 0.5 mm in diameter
2. Post-operative edema is minimal.
3. Scarring is less.
4. Healing is rapid due to minimal damage to the surrounding structures.



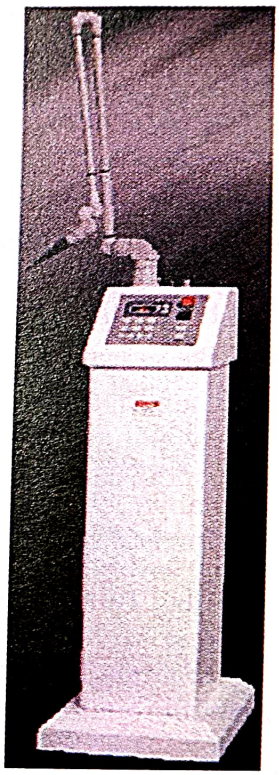
Effects of laser beam

Disadvantages

1. Cost of therapy
2. Biopsy may not be possible, as tissue is vapourised
3. Precautions to be taken by staff during its use.

Precautions during use of LASER:

1. Protective eye glasses are worn by the staff as well as patients eyes are protected.
2. Anaesthetic gases used should be only the non- inflammatory ones.
3. Smoke evacuation done using suction.
4. Exposed areas are protected by covering them with soaked towels or pads
5. Endotracheal tube is wrapped with a metallic foil.



CO_2 LASER machine

CHAPTER 65

Cryosurgery

It is the rapid destruction of tissues by using a probe that produces temperatures below - 30° C.

Mechanism of Action

The rapid freezing of tissues and their slow thawing causes tissue destruction and this fact is used to treat various lesions, including neoplasms.

1. Denaturataion of proteins of the cell membrane increases its permeablilty to cations and on thawing, these cells engorged with cations undergo lysis.
2. Intracellular dehydration occurs due to ice crystallisation.
3. Cell membrane rupture.
4. Enzymatic inhibition arrests the respiratory function of the cell.
5. Thermal shock
6. Cryo immunisation.
7. Local ischaemia and microthrombosis of blood vessels occur.

Technique :

Freezing can be performed by direct application of the gases or through cryoprobe. Cryoprobe works on the principle of Joule Thompson, which states that cooling is produced when a rapidly expanding gas passes through a narrow orifice or a liquified gas becomes gaseous at the tip of the probe. Nitrous oxide cryoprobe can achieve a temperature of –70 C while liquified nitrogen can cool upto –196 C. Direct nitrogen spray may be used for freezing large tumours on the skin surface.

A suitable cryoprobe is applied into or upon the tissue and the latter frozen quickly for 3-8 minutes and then allowed to thaw slowly. This procedure is repeated once or twice and area frozen should include margin of the tissue. There is hyperaemia followed by inflammation. Necrosis sets in with slough formation. The necrotic slough falls of in 3-6 weeks and healthy granulation tissue covers the tissues which heal by secondary intention. Repeat cycles of cryotherapy may be required to achieve the desired result.

Indications

1. **Oral cavity**
   i. Tonsillectomy
   ii. Granular Pharyngitis.
   iii. Epulis
   iv. Palatal ulcers
2. **Malignant tumours** – are given palliative treatment eg. Base tongue tumours, pharyngeal tumours etc.
3. **Benign tumours**
   i. Papilloma
   ii. Angiofibroma
   iii. Hemangioma
   iv. Glomus
4. **Nasal cavity**
   i. Vasomotor rhinitis and hypertrophied turbinates.
   ii. Epistaxis
   iii. Ethmoidal polypi
5. **Ear** – Meniere's disease
6. **Transnasal hypophysectomy**

Advantages

1. Palliative treatment for recurrence of tumours or for metastasis.
2. Useful for patients with bleeding disorders.
3. High risk (for anaesthesia) patients.
4. Minimal post-op pain.
5. Performed as an office procedure.
6. Scarring is minimal.

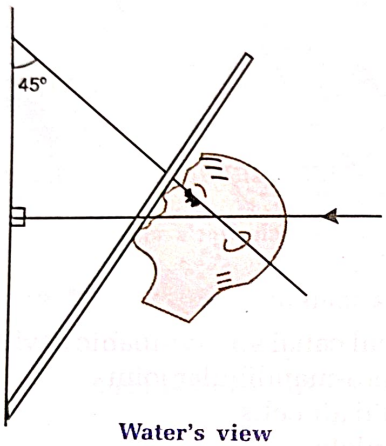
Disadvantages

1. Margins of the tumour cannot be made out.
2. Tissue not available for Histopathology examination.
3. Depigmentation and loss of hair for skin lesions.
4. No control on the depth of freezing.

CHAPTER 66
Radiology

1. Water's view (Occipito-mental)

The patient sits facing the X-ray plate with his nose and chin touching the plate. The head is extended till a position, where the orbito-meatal line makes an angle of 45° with the plate.



Water's view

The various structure seen are:

i. Maxillary sinus
ii. Frontal sinuses projected obliquely
iii. Few ethmoidal cells
iv. Sphenoid sinus through an open mouth
v. The odontoid process just below the mentum confirming adequate extension of the head.

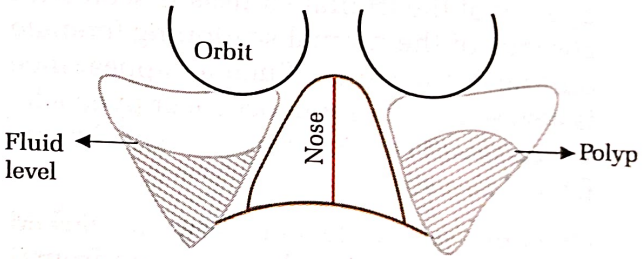
A Water's view can be grouped under one of the following headings:

(a) ***Normal*** – The odontoid process lies exactly below the mentum. The maxillary sinuses are seen as radiolucent cavities. To label a maxillary sinus as pathological, requires it to be more radio-opaque as compared to the orbit.

(b) ***Maxillary sinusitis*** – Haziness (radio-opacity) of the sinus is seen. The mucosa may show a thickening in chronic sinusitis. A fluid level is seen sometimes which has concavity pointing upwards. The final confirmation of fluid level is done by repeating the plate in the lying down position, resulting in obliteration of the fluid level.

(c) ***Polyp*** – Opacity is seen as compared to the rest of the maxillary sinus. Here, there is a convexity which points upwards and a repeat x-ray plate in lying position shows no shift in opacity.

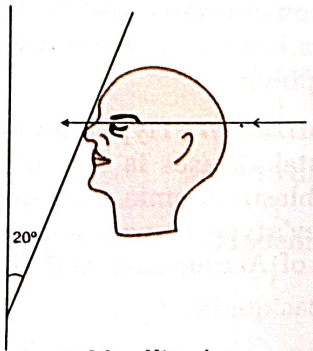
(d) ***Malignancy*** – Opacity of the sinus is seen. Destruction of the walls of the maxillary sinus is diagnostic of malignancy. The distance between the anterolateral wall of maxilla to the coronoid process of the mandible is measured. Increased distance on one side suggests involvement of Infratemporal Fossa by tumour (Handousa's sign).



Water's view

2. Caldwell's view (Occipito-frontal)

The patient is sitting up with his nose and forehead touching the X-ray plate and the X-ray beam makes an angle of 20° with the orbito-meatal line.



Caldwell's view

The various structures are :

i. Frontal sinus
ii. Ethmoid sinus
iii. Orbit
iv. Nasal septum

A Caldwell's view can be grouped under one of the following headings:

a. ***Normal*** – There is no such thing as a normal frontal sinus from the anatomical view point, as there is no constant pattern of pneumatisation. Therefore there is not only individual variation in size and shape but also asymmetry seen of the paired sinuses, this should not be taken as pathological. Normally the frontal sinus shows a radiolucent shadow and individual cells are made out, giving it a scalloping (clove like) appearance. Kartagener's syndrome is a triad of aplasia of the frontal sinuses, bronchiectasis and dextrocardia.

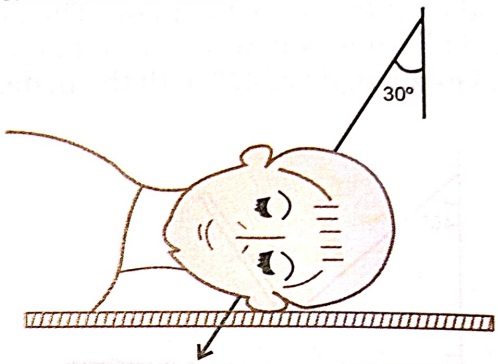
b. ***Chronic Frontal Sinusitis*** – Increased opacity of the frontal sinuses is seen with absence of the normal scalloping (crenate outline of the sinus). Such an appearance is also seen with a mucocoele or pyocoele, which results as a complication of chronic frontal sinusitis.

c. ***Osteoma*** – This lesion shows a marked opacity (denser than bone) in the frontal sinus. The patient is usually asymptomatic and the finding coincidental. The removal of a osteoma is decided only after an X-ray lateral skull is taken which gives an idea of the exact extent of the osteoma. Gardener's syndrome is a traid, which is inherited as an autosomal dominant trait consisting of osteomas, soft-tissue masses and multiple colonic polypi.

d. ***Pneumatization*** – Hypopneumatization of the frontal sinuses is common in severe Erythroblastic anemia, whether Sickle cell or Cooley's type. Hyperpneumatization is a feature of Acromegaly and Sturge-Weber disease.

3. Schuller's view (Mastoid)

The two sides are taken separately. Patient lying in the lateral position and the side to be examined lies in contact with the X-ray plate. The central ray is directed at an angulation of 30° to prevent overlap of the other mastoid bone. This angulation of the X-ray beam makes it different from the lateral-skull view.



Schuller's view

Structures seen are :

i. External canal and tympanic cavity
ii. Temporo-mandibular joint
iii. Mastoid air cells
iv. Dural plate
v. Sinus plate
vi. Dense bone of the labyrinth.

Types of Mastoid:

(a) ***Pneumatic*** – Air cells are seen not only covering the mastoid but also beyond the limits of the dural plate and sinus plate.

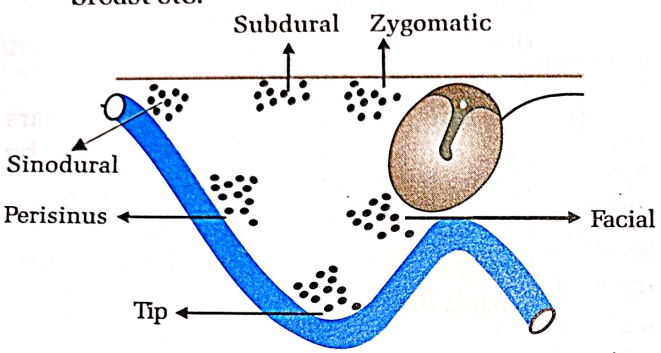
(b) ***Moderate*** – The air cells are seen filling up the mastoid cavity, however do not cross the confines of the dural plate and sinus plate.

(c) ***Sclerotic*** – Absence of air cells is notable. The whole mastoid appears to be small in size with marked opacity. This is common feature in chronic otitis media but however sometimes also seen in normal patients.

(d) ***Mastoid with a Radiolucent cavity*** – Here a single radiolucent shadow is seen in an otherwise sclerotic mastoid. Even though it indicates disease, it may also be seen in normal patients. Just from the X-ray it is impossible to spot out the exact diagnosis.

Causes

i. Cholesteatoma
ii. Operated mastoidectomy
iii. Large Antral cell
iv. Large Peri-antral cell
v. Malignancy
vi. Chronic mastoiditis with granulations
vii. Eosinophilic granuloma
viii. Tuberculosis
ix. Multiple Myeloma
x. Skull metastasis from kidney, bronchus, breast etc.



Right Mastoid cells

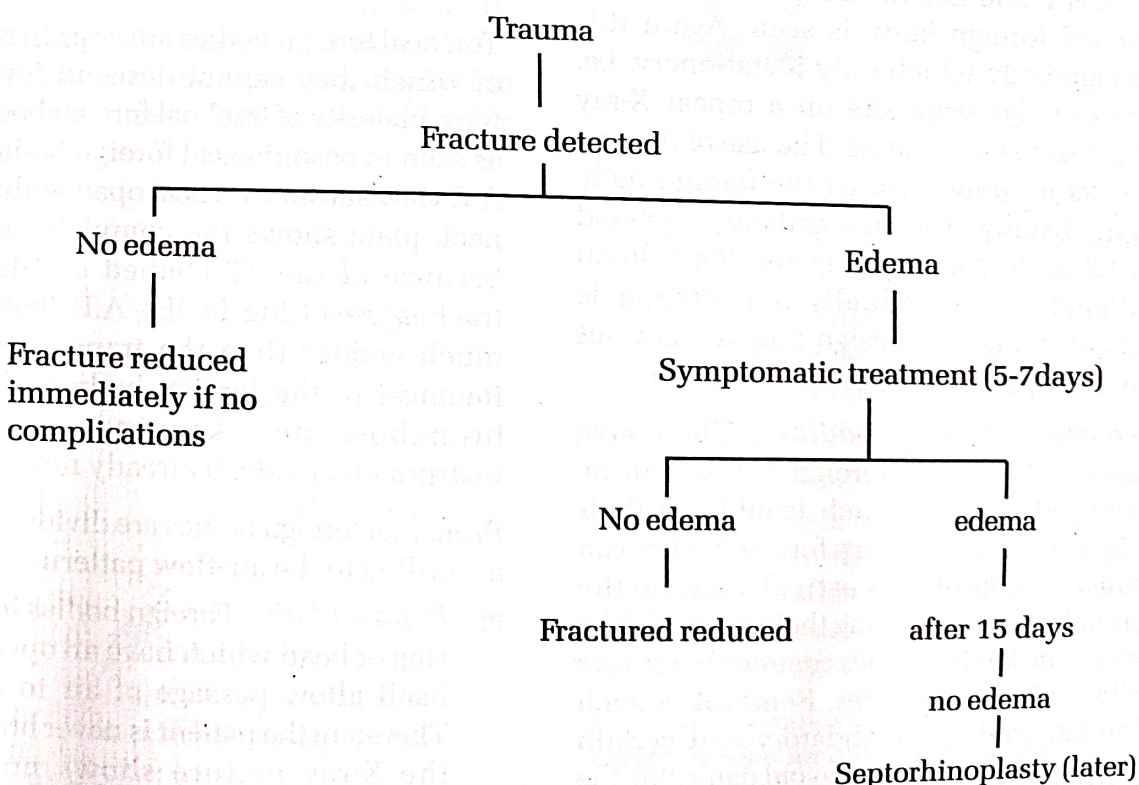
4. Fracture Nasal bones

The patient sits erect with the head in the lateral position and the X-rays are directed to the root of the nose. Any disruption seen in the nasal bones architecture would indicate a fracture of the nasal bones.

Reduction of this fracture is done only as an elective procedure, i.e. for cosmetic reasons. Before doing so, the complications caused by fractured nasal bones are ruled out. They are:

i. CSF rhinorrhoea
ii. Epistaxis
iii. Septal hematoma
iv. Proptosis
v. Edema (which would mask the result of reduction).

Time of reduction fracture :



5. Oesophageal Foreign bodies

The commonest foreign bodies lodged in the oesophagus are coins, marbles or traumatic F.B. like a denture, pin or meat bone.

a. ***Coin*** – They lodge in the oesophagus occupying a classical position that is in the transverse plane. This is due to the fact, that the transverse diameter is much greater than the longitudinal diameter. Therefore, on an A.P. view of the neck-chest, the whole coin is seen showing a totally radio-opaque shadow. A lateral plate is also taken to confirm the exact position of the foreign body and also to rule out second foreign body if overlapping the first. On a lateral plate a vertical slit like structure is seen. A foreign body which remains stationery for at least 6 hours is removed by Oesophagoscopy done under General Anaesthesia.

b. ***Marble*** – This also shows a radio-opaque shadow which is less dense as compared to a coin. The other feature being that on both A-P and Lateral X-ray film a circular shaped foreign body is seen. Again this foreign body which only if stationery, i.e. stays at the same site on a repeat X-ray requires to be removed. The use of forceps results in, only pushing the foreign body more distally. A Foley's catheter is passed distal to the foreign body and the balloon inflated. Now, gradually the catheter is removed and the foreign body comes out taking support of the inflated balloon.

c. ***Traumatic foreign bodies*** – The above described types of foreign bodies can be removed without much trouble as their edges are smooth. Sharp foreign bodies can cause a complete vertical tear in the oesophageal wall during their removal. The traumatic foreign bodies commonly seen are either pins or dentures. Removal of such foreign bodies is mandatory and certain precautions are taken to avoid damaging the oesophagus. For example an open safety-pin is removed using one of the following manoeuvres.

i. The pointed end is taken into the oesophagoscope and the foreign body removed.
ii. If the tip of the traumatic part is pointing downwards, it can be removed in the same position.
iii. The foreign body may be pushed downward in the stomach and rotated such that the sharp end points downward. Following this it is removed without any difficulty.
iv. The foreign body can be cut with shears and each small piece is removed by taking it within the oesophagoscope.
v. The open safety-pin can be closed by using a safety-pin closing forceps, after which it is removed.

6. Tracheo-Bronchial foreign bodies

These foreign bodies may lead to a life threatening situation due to the obstruction caused in the respiratory passage.

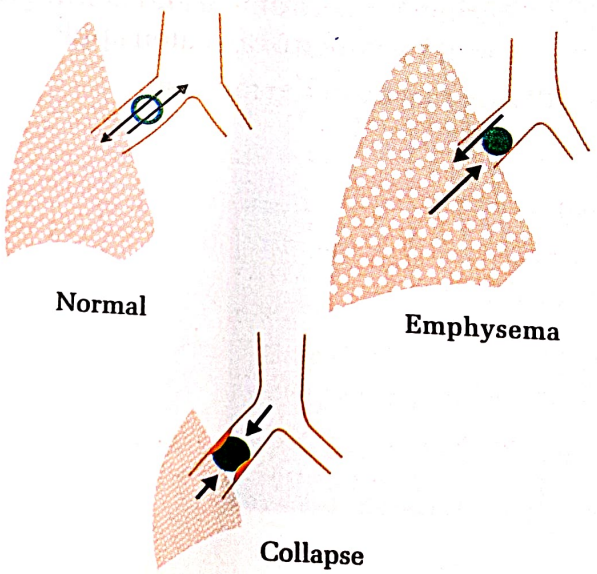
Tracheal foreign bodies are large in size because of which they cannot descend further. Coins form majority of tracheal foreign bodies. Unlike as seen in oesophageal foreign bodies, here the A.P. view shows a vertical opaque slit and lateral neck plate shows the complete coin. This is because of the 'C' shaped cartilages of the trachea, resulting in the A.P. diameter to be much greater than the transverse diameter. Removal of the foreign body is done with a bronchoscope, keeping tracheostomy instruments ready, if already not done.

Bronchial foreign bodies are divided into 3 types according to the air-flow pattern.

a) ***Bypass of air*** – Foreign bodies like a button, ring or bead which have an opening within itself allow passage of air to either side. Therefore the patient is never breathless and the X-ray picture shows no abnormal findings other than the foreign body.

b) ***One way airway obstruction*** – Metallic and other non-organic foreign bodies lead to an unidirectional obstruction in the airway.On inspiration the bronchial diameter increases and this leads to passage of air distal to the foreign body. However on expiration as the bronchial diameter decreases, it leads to entrapment of air distal to the foreign body. This change in airway pattern leads to obstructive emphysema and therefore the X-ray findings and breathlessness.

c) ***Total airway obstruction*** – Vegetable foreign bodies like a pea or groundnut, swell up in the bronchus due to their hygroscopic nature. This increase in size of the foreign body cause impaction of the foreign body onto the bronchial wall. The result being that air cannot pass distal to the foreign body, nor can it escape out. This leads to a collapse of the lung segment distal to the foreign body and compensatory emphysema of the remaining one. Changes occur accordingly which are seen on the X-ray. The classical feature is ***Mediastinal shift***: which is the change in position of the mediastinum with each phase of respiration on account of the collapse caused by the foreign body.



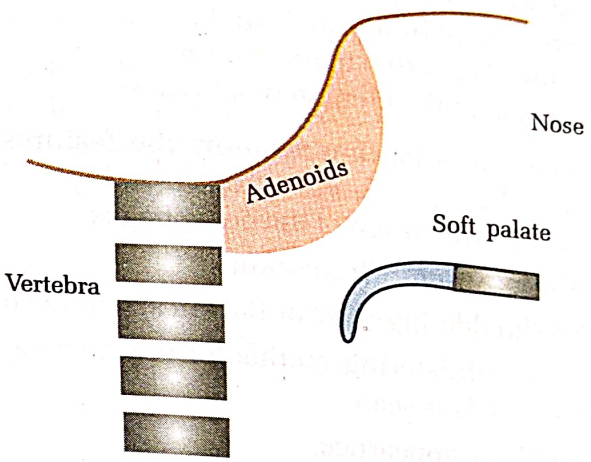
X-ray findings with different types of foreign bodies

7. Lateral Skull (Nasopharynx)

Normally the nasopharynx is seen as a radiolucent shadow because it is occupied by air.

i. Shift of the air shadow posteriorly occurs with an antrochoanal polyp, as it arises from anteriorly. The air shadow is reduced to a small outlining shadow around the polyp (*crescent sign*).

iii. Shift of the radiolucent shadow anteriorly is seen *with Adenoids (children)* and *Nasopharyngeal carcinoma (elderly)*, both of them arising posteriorly.



X-ray Nasopharynx

8. Lateral neck (Retropharyngeal – abscess)

The diagnosis of a Retropharyngeal abscess is made by :

i) Marked *increase in the prevertebral space* area, that is it being more than three-fourths of the size of body of the vertebra.

ii) *Air-fluid level.* Normally the pharynx is a collapsed structure, not containing any air.

iii) *Loss of normal curvature of the spine* leads to straightening of the cervical spine. This is due to reflex spasm of the prevertebral muscles occuring secondary to the retropharyngeal abscess.

Once the diagnosis made, the vertebrae are carefully seen for any destruction and presence of any foreign body (mutton bone) is to be ruled

out. The commonest cause of retropharyngeal abscess is Koch's spine and foreign body in the pharynx (adults); dental and tonsillar infections (infants). Drainage of the abscess which is done under local anaesthesia (whatever the age of the patient), injectable antibiotics, intravenous fluids and treatment of the cause if detected.

9. Achalasia Cardia

Here a plain X-ray is more informative than even a Barium swallow. The classical features seen are:

i) Presence of an air-fluid level
ii) Regular dilatation of the oesophagus (sigmoid oesophagus)
iii) Absence of fundic air bubble
iv) Lung abcess due to aspiration
v) Mediastinal widening in late cases.

However, on a barium swallow the features noted are:

i) Regular dilatation of the oesophagus
ii) Abrupt stricture formation
iii) No shouldering effect or Rat tail appearance
iv) On administering methacholine, reverse peristalsis is seen.
v) Bird beak appearnce.

10. Oesophageal Strictures:

The stricture of the oesophagus on a barium swallow is classified as a benign or malignant type.

I. Benign stricture:

i) Multiple in number
ii) Usually at the sites of normal constrictions of the oesophagus
iii) Mucosal regularity is maintained
iv) Marked proximal dilatation of the oesophagus.

II. Malignant stricture:

i) Usually single
ii) Found at any site in the oesophagus
iii) No proximal dilatation seen because of submucosal infiltrations by the malignant cells. In a malignant stricture growth is very fast and proximal dilatation occurs only after some time
iv) *'Shouldering effect'* is seen due to everted margins of a malignant ulcer causing an acute angle of the barium outline.
v) *'Rat Tail'* appearance is seen as the oesophageal lumen becomes very narrow and only a thin streak of Barium can pass through it.
vi) *'Apple-core appearance'*. Extremely narrow stricture of the oesophagus with normal mucosa above and below gives the appearance of an eaten apple
vii) Irregularity of the mucosa.

CHAPTER 67

CT Scan

It is a non- invasive radiological method used for diagnosing bony and soft tissue diseases of the body, in inaccessible areas which cannot be visualised by the conventional methods of radiology.

Indications:

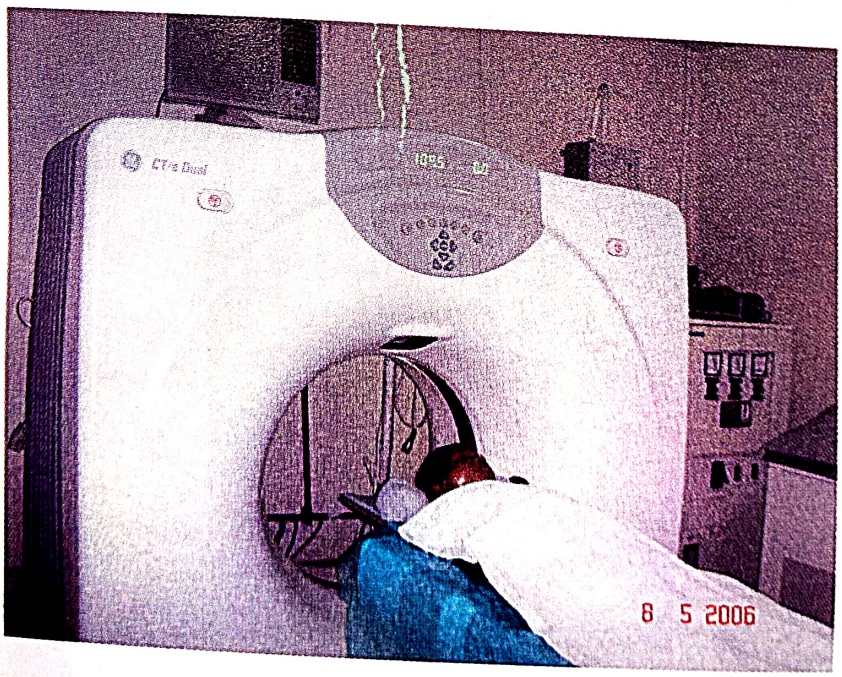
1. **Ear**
   - i) Acoustic Neuroma
   - ii) Cholesteatoma
   - iii) Ossicular malformation
   - iv) Fractures
2. **Paranasal Sinus**
   - i) Tumours
   - ii) Sinusitis
3. **Orbit**
   - i) Extension of Tumours
   - ii) Optic nerve pathway
   - iii) Retro-orbital pathology
4. **Cerebellopontine Angle**
   - i) Tumours
   - ii) Hemorrhage
   - iii) Infarction

Disadvantages

- i) Poor soft tissue scanning
- ii) Expensive

Advantages

- i) Detects early lesion
- ii) Non-invasive technique
- iii) Contrast enhancement possible
- iv) Hardly any radiation exposure.



CT Scan machine